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Book Reviews.

Other Books Received.

Index.

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CONTENTS

ROBERT H. THOULESS. Effect of prejudice on reasoning.

H. TAJFEL. The anchoring effects of value in a scale of judgements.

J. R. SMYTHIES. The stroboscopic patterns. II. The phenomenology of the bright phase and after-images.

PAUL BAKAN. Extraversion-introversion and improvement in an auditory vigilance task.

N. O'CONNOR and J. P. DAS. Lability in schizophrenia.

PETER H. GLOW. The blocking effect of benactyzine hydrochloride on a behaviour disturbance induced with lysergic acid diethylamide.

R. CONRAD. Errors of immediate memory.

H. J. EYSENCK. Serial position effects in nonsense syllable learning as a function of interlist rest pauses.

JEROME S. BRUNER. Inhelder and Piaget's THE GROWTH OF LOGICAL THINKING. I. A psychologist's viewpoint.

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THE QUARTERLY JOURNAL OF
EXPERIMENTAL PSYCHOLOGY

Vol. XI 1959 Part 4

EVIDENCE OF A SINGLE-CHANNEL DECISION MECHANISM LIMITING PERFORMANCE IN A SERIAL REACTION TASK*

BY

A. T. WELFORD

From the Psychological Laboratory, Cambridge

An experiment is described in which the subject sat facing a display of two neon bulbs. When the left-hand bulb lit he pressed a key under his left hand, and when the right hand bulb lit he pressed a key under his right hand. The left-hand bulb gave brief flashes at random intervals averaging about 4 sec. The right-hand bulb gave a brief flash at regular intervals of about 4 sec.

The experiment repeats (the author believes for the first time) certain essential conditions of Vince's (1948, 1950) experiments and, following detailed scrutiny of every pair of responses, is taken as evidence for the following statements:—

(*a*) The response to a signal arriving during the reaction time to a former signal will be delayed by an amount approximately equal to the time elapsing between the arrival of the signal and the end of the reaction time to the former signal.

(*b*) An exception to this may occur when two signals arrive close together. In this case the two signals may be responded to as a single group.

(*c*) Delays can be occasioned by the monitoring of responses as well as by reactions to signals.

(*d*) "Grouping" of signal and monitoring may occur when a signal arrives close to the beginning of the movement made in response to a previous signal.

A survey is made of current theories in this field and suggestions given for further research.

Introduction

The experiment to be described here was suggested by a conflict of fact between the work of Vince (1948, 1950) and of Davis (1956). Both Vince and Davis showed that if one signal for action (S2) followed closely upon another (S1), the response to S2 was likely to be longer than was normal for a signal well separated from others in time. Davis's results indicated that delay occurred when S2 arrived during a period about equal to the reaction time of S1 (i.e. TR1), but not if it arrived later. Vince's results, however, showed that substantial delays also occurred when S2 arrived during or shortly after the movement (M1) made in response to the previous signal. The facts ascertained by Vince were incorporated, along with others in a theory (Welford, 1952) that the delays were due to a "single channel" somewhere in the subject's central mechanisms which could be occupied either by dealing with

* Based on a Contribution to the Symposium on "The Psychological Refractory Period" held at the EPS Meeting in London, January, 1959.

signals from the outside or by monitoring the subject's responding movements—a process which Hick (1948) suggested would almost inevitably occur. It seemed that the channel dealt with "items" in the order of their arrival to the extent that if S2 arrived before the end of TR1, it was dealt with as soon as TR1 had ended, but that if M1 had started before S2 appeared some monitoring of M1 had to be completed before the single channel began to "process" the information from S2.

Davis's conclusion that no delays occurred when S2 arrived after the end of TR1 has been supported by Marill (1957). Vince's results have been supported by Davis's own later work (1957), by Elithorn and Lawrence (1955) and by Fraisse 1957). The interpretation of all these results is in some doubt, however, because if delays occur due to the monitoring of M1, we should expect them to bear some relation to its duration, but this, since Vince's work, has never been measured. Interpretation and analysis have, indeed, been excessively difficult in all the work previously reported, because much of the theory has been couched in terms of the time at which S2 arrived in relation to TR1 or M1, but the results have been analysed in terms of the time between S1 and S2. If TR1 is variable in length, as it always is, there is thus a zone of intervals between S1 and S2 over which it is uncertain whether S2 arrived before or after the end of TR1.

A further possible complication arises because Vince used a continuous task in which a whole series of signals were presented at various intervals, but subsequent work has all been done with discrete pairs of signals. Davis and Fraisse have ingeniously avoided the pitfall that when the subject does not know which of the two signals will be S1, the giving of S1 sharply reduces the information conveyed by S2 and thus makes the TR2 that would be expected if there was no delay different from the observed TR1. Nevertheless the lurking suspicion remains that the subject may be able to achieve a performance with discrete pairs of signals that he cannot maintain with a continuous series. For example, he may, if he knows he is only going to have two signals, be able to defer monitoring his responses until both signals have been dealt with.

Method

In the present experiment the subject sat at a table. Opposite him, about 2½ ft. away, was a vertical board through holes in which appeared two small neon bulbs 6 in. apart. Under his hands were two morse keys. He was instructed that when the left-hand neon lit he was to press the left-hand key and when the right-hand neon lit he was to press the right-hand key, *in each case as soon as possible.*

The neons flashed for approximately 200 millisec. The left flashed at intervals determined by an electronic random-interval generator, at an average rate of a little more than one flash per 4 sec. The right-hand neon flashed at regular intervals of about 4 sec. It had originally been hoped to use two random interval generators but a second could not be obtained at the time. As it turned out this fact did not seem to be a serious disadvantage: the subjects did not appear to be aware, or at any rate were not acutely so, that the right-hand bulb lit at regular intervals, and the reaction times to both right-hand and left-hand lights were, on average, very similar.

Five subjects were used with ages ranging from 20 to 41. Each performed for a single continuous run lasting about 20 min.

The times of the appearance of each signal and of the beginning and end of each responding movement were recorded by a SETAR electronic digital recorder to the nearest 0·01 sec. (N. T. Welford, 1952).

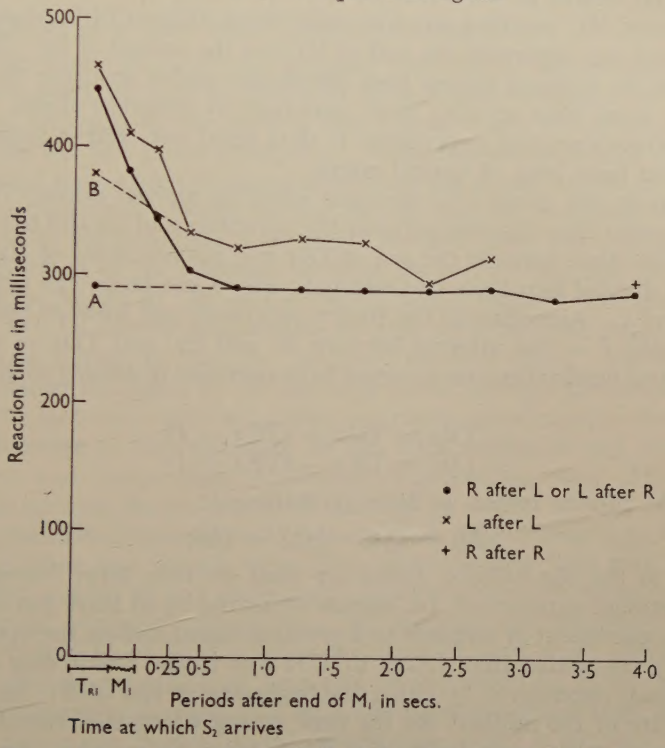
Discussion of Results

When calculating the results all signals were divided into categories (*a*) according to whether or not they appeared on the same side as the preceding signal, and (*b*) according to their relation in time to the response to the previous signal. The

average reaction times are shown in Figure 1. The data on which this is based do not include the few cases in which an error was made, for example by pressing the wrong key. The categories of "left after right" and "right after left" were combined because the mean reaction times were very similar. The means shown are for the readings of all subjects taken together in spite of the fact that, owing to the conditions of the experiment and the manner of classifying the results, the proportions of readings from the different subjects in the various categories were not equal.

FIGURE 1

Mean Reaction Times to signals following at different intervals after previous signals



The points at the extreme left of the two curves are for signals which arrive during the reaction time to a previous signal. The points immediately to the right of these are for signals which arrive during the execution of a movement in response to a previous signal. Subsequent points are for signals which arrive at various intervals after the end of a movement made in response to a previous signal. The divisions between the ranges of interval are indicated, to scale, on the abscissa.

The mean TR2 when S2 came during TR1 or M1 or within 250 millisec. of the end of M1 was significantly different from the mean reaction time when S2 came 500 millisec. or more after the end of M1. In each case P was less than 0·001.

Presentation of the results in this way was felt to be justified, however, following three checks:—

(i) The effects to be discussed were shown, with some minor irregularities attributable to the small numbers of readings, by each of the five subjects.

(ii) The means of the individual subjects' means were closely similar to those shown.

(iii) Every individual pair of readings was scrutinized to see whether it could adequately be accounted for in terms of the theory to be put forward. This detailed scrutiny was one of the original objects of the experiment and provides a very much more rigorous test of theory than does a consideration of mean results alone.

A. *Overall results for successive signals on different sides*

These results are shown by the heavy line in Figure 1 and indicate that there were substantial delays to M2 when S2 arrived during TR1 or during or shortly after the recorded M1, but that mean reaction times differed little when an interval of more than 0·5 sec. separated the end of M1 and the arrival of S2. There can be no question of the subjects having been physically unable to make the two movements at the same time as they were executed by different limbs. A possible objection to Vince's original experiment is thus ruled out, and it seems clear that the delays must have been of central origin.

We can divide the mean TR2 for cases when S2 arrived during TR1 into two parts: (i) the mean time elapsing between the appearance of S2 and the end of TR1, and (ii) the mean time between the end of TR1 and the beginning of M2. The total TR2 is shown divided into these two intervals, above and below the point A respectively in Figure 1. According to the theory previously put forward (Welford, 1952) we have, writing I = the interval between S1 and S2, and TD2 = the time for which the central mechanisms are assumed to be occupied in dealing with the information from S2:

$$\mathrm{TR2} = \mathrm{TD2} + (\mathrm{TR1} - I)$$
$$\therefore \mathrm{TD2} = \mathrm{TR2} - (\mathrm{TR1} - I)$$

In terms of the present means we have (in millisec.):

$$289 = 451 - (324 - 162)$$

This is close to the 282 millisec. found for what we may term "*normal*" reaction times in the present experiment, i.e. signals separated by at least 500 millisec. from the end of the movement in response to a previous signal and on the opposite side.

The theory postulated that delays to TD2 due to the monitoring of M1 might arise from signals engendered by either the beginning or end of M1, and tentatively assigned a figure of 150 millisec. for the time during which the central mechanisms were so occupied. We may denote the times taken to monitor signals from the beginning and the end of a movement as TF*b* and TF*e* respectively. We cannot calculate these times accurately from the data, but a rough approximation to TF*b* may be calculated from the equation

$$\mathrm{TF}b = \mathrm{TR2} + I' - \mathrm{TD2}$$

where I' = the interval between the beginning of M1 and the arrival of S2. Taking TD2 = 289 millisec. as calculated above we have:

$$171 = 381 + 79 - 289.$$

This figure agrees reasonably well with the 150 millisec. inferred from Vince's and other results.

A method of arriving at a figure for TF*e* is described in Appendix B. No exact quantity can be calculated but it seems likely to have lain between 143 and 199 millisec.—again a tolerably fair agreement with the 150 millisec. previously postulated.

B. *Overall results for successive signals on the same side*

Mean reaction times when S2 was on the same side as S1 were in all cases longer than when S1 and S2 were on opposite sides. With very short *I* the lengthening of TR2 could be in part due to the necessity of completing M1 before beginning M2, and the point B in Figure 1, calculated in a similar manner to the point A, shows a TD2 substantially greater than a "normal" reaction time. This can hardly be the explanation, however, of longer reaction times when there was an interval of a second or more between the end of M1 and the arrival of S2. In these cases it seems reasonable to assume a delay due to an "expectancy effect" such as those studied by Mowrer (1940). The subjects seem to have tended to expect successive signals to be on opposite sides.

C. *Detailed scrutiny of cases where S2 arrived during TR1*

The results shown in Figure 1, especially those for S1 and S2 on opposite sides, seemed almost too good a fit to theory to be true, and subsequent scrutiny showed this to be so, although it also showed that Figure 1 was not seriously misleading. In a few cases, M2 was made before M1. These reversed reactions were obviously exceptional and were excluded when calculating the means shown in Figure 1. In other cases the interval between the end of TR1 and the beginning of M2, was too short for it to be assumed that a "normal" reaction time had elaspsed.

The distribution of the intervals between the end of TR1 and the beginning of M2 is shown in Figure 2. If, as the theory would require, this distribution is of times taken by the central mechanisms to deal with the data from S2, we should expect it to be similar to that for "normal" reaction times. The distribution of these is also shown in Figure 2. It will be at once clear that the former distribution is wider. The fact that there are rather too many occurrences in the 350 millisec. and upwards range is not important as the total number is not large and many chance factors may cause slight lengthening of a reaction time. The more serious problem is to account for the values which are too small.

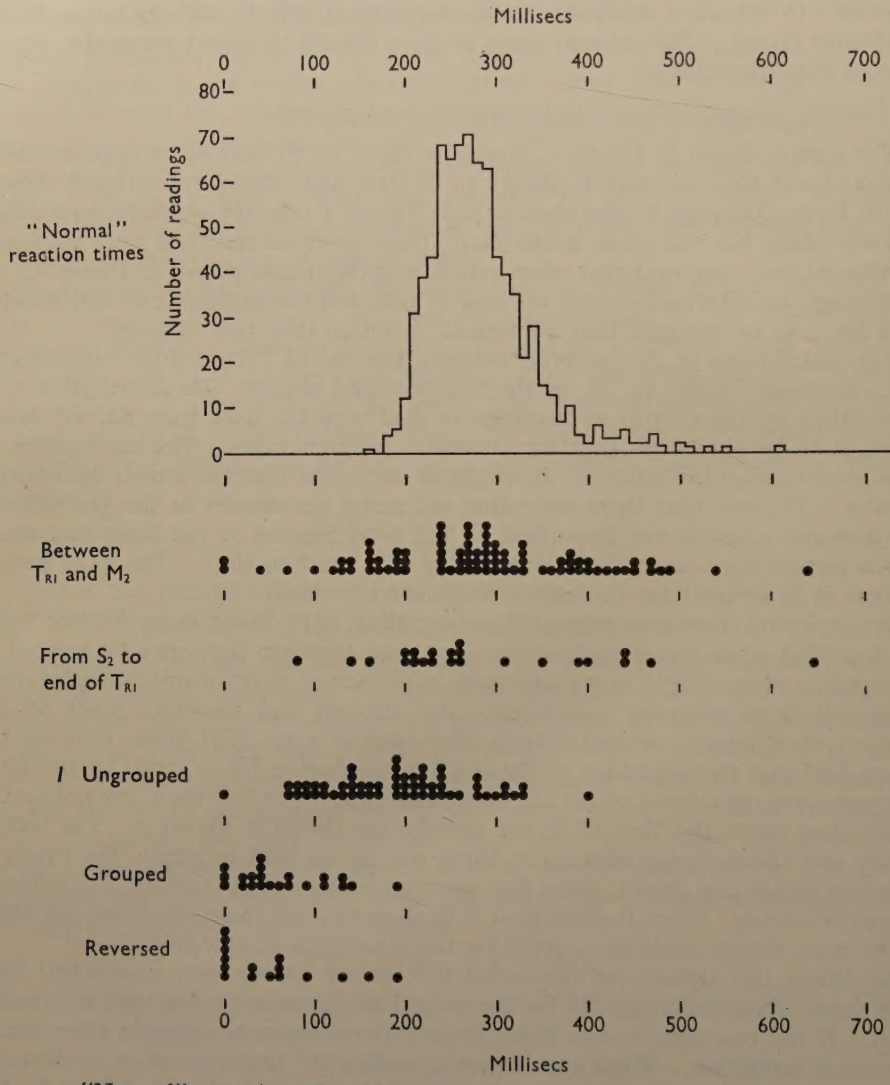
It has several times been suggested (see e.g. Hick, 1948, Vince, 1949, Welford, 1952, pp. 8–9) that when two signals occur very close together they may be treated as "grouped" and reacted to as a single unit—a process of which many examples could be quoted from everyday perception. An attempt was therefore made to see whether, according to various criteria, the cases of very short times between the end of TR1 and the beginning of M2 could be accounted for in terms of grouping. All cases were considered where the time was equal to or less than 200 millisec.—i.e. all cases below the "break" in the distribution shown in Figure 2. The several criteria and the evidence relevant to them will be outlined in turn. The evidence cannot be said in any sense to *prove* that grouping took place or to distinguish between different theories. What is attempted is to show that *all* cases which are not fitted by the main theory could be covered by the hypothesis of grouping.

(*a*) When two signals are responded to together as one unit a sufficient time must elapse between S2 and M1 for the central mechanisms to deal with the double signal. If the two signals were being dealt with on separate channels there would be no such limitation. What constitutes a "sufficient" time cannot on evidence at present available be specified with certainty. We should not expect it to be the equivalent of two "normal" reaction times. Since the double response would be relatively rare we might perhaps expect the time to be longer than one "normal" time; on the other hand since both movements have to be made there is no longer a need to choose accurately which to make and thus the time might sometimes be shorter than a "normal" reaction time provided a few reversals of the correct order

were permissible. From Hick's (1952) formulation that $T R = K \log (n + 1)$ where n is the number of equiprobable choices and K is a constant, we should expect the time from S2 to M1 to be distributed with a mean not less than 1·58/2 of a "normal" reaction time, i.e. as for a simple instead of a two-choice reaction. The observed

FIGURE 2

Distribution of "Normal" Reaction Times and of certain quantities for cases in which S2 arrived during TR1



"Normal" reaction times are those in which S1 and S2 were on different sides and S2 arrived 500 millisec. or more after the end of M1. Cases in which a further S arrived during TR1 were excluded.

Owing to a limitation in the recording system intervals of 0 and 10 millisec. were not distinguished, all being recorded as 0. Allowance for half the zero readings having been in fact 10 millisec. has been made when calculating the means shown in Table I and Appendix A.

times, shown in Figure 2 are all reasonable on this assumption except the one indicated on the extreme left. This, however, was for a case in which the interval between TR1 and M2 was 200 millisec. and we may suppose that this particular response was not grouped.

(*b*) Grouped responses would, on the view outlined, be likely to occur when I was very short, although they might occur when the interval was longer if the subject's attention were momentarily deflected when S1 appeared. Distributions given in Figure 2 show that short times between TR1 and M2 were strikingly associated with short I, and indeed approached those for the cases in which the responses were reversed. Reversal should probably be regarded as a sign of grouping, at least in many cases, and so the similarity of the two distributions is not surprising.

(*c*) It might perhaps be expected that if responses were grouped they would be made simultaneously. This, however, occurred only rarely. The movements in most of the grouped pairs of reactions were successive although often M2 would begin before M1 had finished. Introspectively it seemed that although two signals might be perceived "together" they were nevertheless recognized as successive and that there was a compelling tendency to reproduce the successiveness in the responses. Manifestly the actual time interval between the responses was not reproduced.

(*d*) Since the time taken to deal with the data from S1 and S2 together is assumed to be less than is required if they are dealt with serially, we should expect grouping to result in shorter TR2. The mean TR2 for the grouped responses was, as shown in Appendix A, shorter than that for ungrouped responses, although it was not significantly so. Little weight can be placed upon this finding, however, as it is really tautologous: the short interval between the end of TR1 and the beginning of M2 (which was the original ground for suspecting grouping) might merely have been the result of short TR2.

If, however, grouping is generally associated with short TR2 and also with short I we should expect that the mean TR2 for *all* pairs in which I is very short should be less than for pairs in which I is somewhat longer. The range of I over which this will hold is limited since when S2 came late during TR1, TR2 would not be much lengthened in ungrouped cases. Mean TR2 was therefore compared for I = 0–60 millisec. and I = 70–150 millisec. with results shown in Table I. If cases are included in which the responses were reversed, the difference of TR2 is substantial and highly significant. If these cases are omitted the difference is smaller and not significant although still in the expected direction. This last result must be viewed against the fact that if all responses had been ungrouped and TD2 had begun only when TR1 had finished, we should have expected TR2 for the I = 0–60 millisec. range to have been *longer* than that for the I = 70–150 millisec. The quantity that would be expected to have remained, on average, constant between the two ranges of I is the time from the appearance of S1 to the beginning of M2, i.e. (TR2 + I). Table I shows the difference of this quantity between the two ranges of I to be clearly significant.

Such tendency as there is for grouped responses to have short TR2 and to be associated with short I would seem a likely explanation of the fact noted by Elithorn and Lawrence (1955) that TR2 was, on average, shorter when I = 20 millisec. than when I = 100 millisec.

(*e*) If it is true, as suggested earlier, that the time taken to deal with two grouped signals may sometimes be longer and sometimes shorter than a "normal" two-choice reaction time, we cannot make any confident prediction about the effects of grouping on the mean TR1, but we should expect an increase of variance. Some lengthening of mean TR1 would be expected if all cases in which S2 arrived during

TR1 are considered, merely because there is a greater chance of this happening during a long TR1. This objection does not apply however if only those cases are taken in which I is less than the minimum "normal" reaction time. Mean and standard deviation of TR1 for $I \leqslant 150$ millisec. are shown in Table II. It can be seen that if cases in which the responses were reversed are included there was a substantial rise of mean TR1. Where these cases are excluded the mean TR1 is still a little longer than "normal" and the variance is still significantly larger.

TABLE I

TR2 FOR CASES IN WHICH S2 ARRIVED DURING TR1
All times in millisec.

	I = 0–60	I = 70–150	*Significance of difference between means*	
			t	$P<$
Including reversed cases:				
Number of readings ..	29	29		
Mean TR2	377	465	2·99	0·01
Excluding reversed cases:				
Number of readings	17	28		
Mean TR2	429	465	1·02	N.S.
Mean (TR2 + I)	458	576	3·35	0·01

TABLE II

COMPARISON OF "NORMAL" TR WITH TR1 FOR CASES IN WHICH S2 ARRIVED DURING TR1 AND I = 0–150 MILLISEC.
All times in millisec.

	Number of cases	*Mean*	*Standard deviation*	*Significance of difference of variance from that for "normal" reaction times*	
				Variance ratio	$P<$
"Normal" TR	797	283	56		
TR1 including reversed cases	58	347	163	8·56	0·001
TR1 excluding reversed cases	44	301	104	3·48	0·001

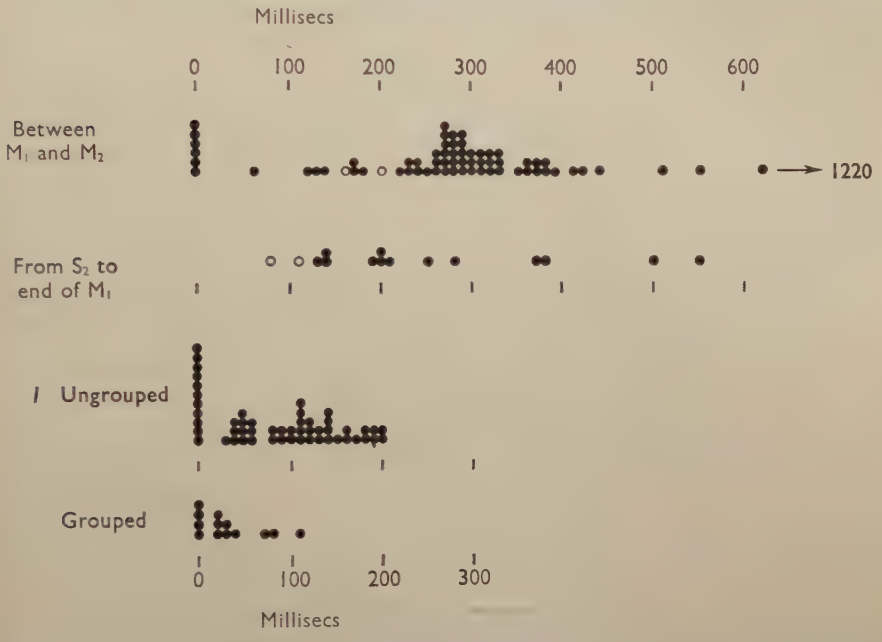
D. *Detailed scrutiny of cases where S2 arrived during M1*

The rough figure of 171 millisec. for TF*b* calculated from the overall results was sufficiently close to the mean first movement time (TM1) of 193 millsec. to suggest that further study might show the two times to be essentially equal. Scrutiny of every case in which S2 appeared during M1 tended to confirm this view provided that due account was taken of occasional cases in which it looked as if grouping had occurred between S2 and the monitoring of M1 similar to that previously indicated in certain cases between S2 and S1.

The times between the end of M1 and the beginning of M2 are plotted in Figure 3. Most of them fall well within the distribution of "normal" reaction times, but again some are far too short. As before, all cases in which the time was 200 millisec. or less were scrutinized for evidence of grouping according to criteria analogous to those used for cases when S2 came during TR1.

FIGURE 3

Distributions of certain quantities for cases in which S2 arrived during M1



The large numbers of zero readings were due to the limitation in the recording system noted in the legend to Figure 2. Allowance had been made in Table IV and Appendix A in the same manner as in Table II.

(*a*) The times from the arrival of S2 until the end of M1 are plotted in Figure 3. The majority fall comfortably within the range of expected values for simple reaction times, i.e. 1·58/2 of those for "normal" reaction times. There are however a few at the left of the distribution which seem suspiciously short. Of these, however, two were associated with times between the end of M1 and the beginning of M2 which would be consistent with the response to S2 not having been grouped with the monitoring of M1, and they have therefore been treated as ungrouped. The corresponding points on the two distributions are distinguished by open circles. A third point in the group was for a pair of reactions in which the onset of S2 coincided with the beginning of M1, and the beginning of M2 coincided with the end of M1. The recorded time—130 millisec.—was thus the duration of both M1 and TR2.

(*b*) By analogy with cases in which S2 came during TR1 we should expect grouping to have been most frequent when the interval I' between the beginning of M1 and the arrival of S2 was short. The distributions in Figure 3 show that this was so.

(*c*) The mean TR2 for all cases, grouped and ungrouped together, in which $I' =$ 0–30 millisec. was found to be lower than that for $I' =$ 40–80 millisec., as shown in

Table 3. The difference was not significant, but it must again be remembered that if all cases had been ungrouped we should have expected TR2 to be *higher* in the former range of I' than in the latter. The quantity expected to be constant would be the time from the beginning of M1 to the beginning of M2, i.e. (TR2 + I'). This is shown for the two ranges of I' in Table III and the difference between the ranges was found to be clearly significant.

TABLE III

TR2 FOR CASES IN WHICH S2 ARRIVED DURING M1

All times in millisec.

	I' = 0–30	I' = 40–80	*Significance of difference between means*	
			t	*P*<
Number of readings	21	12		
Mean TR2	382	427	1·54	N.S.
Mean (TR2 + I')	392	483	3·05	0·01

TABLE IV

COMPARISON OF "NORMAL" M WITH M1 FOR CASES IN WHICH S2 ARRIVED DURING M1, AND I = 0–80 MILLISEC.

All times in millisec.

	Number of cases	*Mean*	*Standard deviation*	*Significance of difference from "normal"*		
				Normal deviate in τ test†	*Variance ratio*	*P*<
"Normal" M*	756	148	59			
M1 in the cases under consideration	39	197		3·38		0·001
			96		2·58	0·001

* "Normal" movement times were those corresponding to the "normal" reaction times in Table II except those cases in which S2 arrived during M1.

† A τ test of the type described by Whitfield (1947) was used owing to the significant difference of variance.

(*d*) The time taken to deal with the information from S2, which is assumed in grouped cases to take place between the onset of S2 and the end of M1, is on average longer than the mean TM for "normal" signals. It would again be tautologous to compare the mean M1 of the grouped cases with that in response to "normal" signals, because, on the criterion of grouping adopted, classification as "grouped" would tend to result from long M1 if M1 and TR2 were independent. Nor again can we compare the TM1 of all cases in which S2 arrived during M1 with the TM of "normal" cases, because the longer TM1 the more chance there is of S2 arriving before it has ended. However, less than 1 per cent. of the recorded "normal" TM were of 80 millisec. or under, so that it was felt safe to

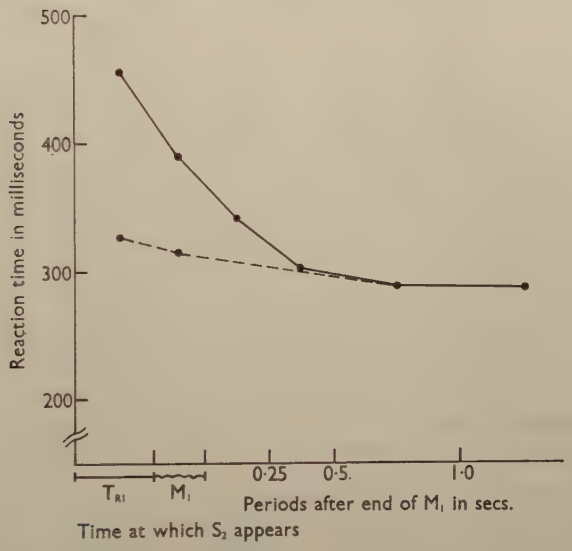
use for comparison all cases in which $I' \leqslant 80$ millisec. The results set out in Table IV show clearly that TM tended to be longer than the "normal" TM, and had a much larger variance. It was also longer, although not quite significantly so, than TM1 in cases where S2 arrived during TR1. It thus seems fairly clear that the arrival of S2 during TR1 had some effect on TM1 in at least a proportion of cases.

E. *Re-examination of the overall results for successive signals on different sides*

The results were then replotted, after the exclusion of all grouped cases, in Figure 4. It can be seen that the calculated times for TD2, taking this to begin at

FIGURE 4

Mean reaction times to ungrouped signals following at different intervals after previous signals



The points on the dotted line are for calculated TD2.

the end of TR1 or M1 as the case may be, show a slight upward curve to the left. What this was due to is not certain, but four possibilities deserve mention:—

(*a*) It may be that there was a residual, "Mowrer effect" with short *I*.

(*b*) The rise could be explained in terms of a central refractory period of constant duration as suggested by Davis (1957).

(*c*) In some of the cases when S2 arrived just before the end of TR1, TR2 was somewhat longer than usual and it seemed possible that grouping of S2 with the monitoring of M1 might have occurred. These cases were not separated from the rest as they were very few and were too unevenly distributed between the subjects for much weight to be given to them. The possibility of such grouping remains, however, and would seem worth exploring in further work. The rise in the case of signals occurring during M1 might perhaps have resulted from a similar grouping with the monitoring of the end of M1, but the evidence was far too slight for this possiblity to be tested.

(*d*) The effect could have been due to some minimal feedback from the beginning of M1 being necessary to "release" the "single channel" from its occupation by data from S1 before it could accept data from an S2 arriving during TR1. In the same way feedback from the end of M1 might have been necessary to release the central mechanisms from their monitoring of M1 and leave them free to deal with data from an S2 arriving during TM1. Such a feedback would introduce a small constant which would delay the beginning of TD2 until after the end of TR1 although it might not, as we shall discuss later, *always* result in an extra delay to M2. Further consideration will be given to this point when the theory is re-examined as a whole.

F. *Conclusions from the present experiment*

The results taken as a whole would seem very fairly consistent with the results of Vince's experiments and with the present author's theoretical formulation although other theories have not been positively excluded. Confirmation of former work is contained in the findings that:—

(*a*) When S2 arrived during TR1 the lengthening of TR2 was in most cases such as to be consistent with the view that a TD2 of the same duration as a "normal" reaction time began at (or very shortly after) the end of TR1.

(*b*) Every case which was not fitted by this theory was consistent with the view that grouping of S1 and S2 had occurred.

(*c*) Lengthening of TR2 was observed in cases in which S2 arrived during or shortly after M1.

In addition, two new points emerge which have not been demonstrated in previous experiments:—

(*d*) When S2 arrives during M1, grouping of S2 with the monitoring of M1 may occur with results analogous to those when S2 arrives during TR1 and is grouped with S1.

(*e*) In cases where S2 arrives during M1 and grouping does *not* occur, the lengthening of TR2 is such as to be consistent with the view that a TD2 of the same duration as a "normal" reaction time begins at (or shortly after) the end of M1.

The Present State of Theory

Looking back over the statements published in this field, we find five types of theory have been advanced to account for the delays in responding to a signal which closely follows a previous signal.

A. The first type, which was responsible for the term "psychological refractory period" postulates that following some event in the chain of processes leading from signal to responding action there is a refractory state, analogous to that found in nerve fibres but of much longer duration—100 to 500 millisec. in different formulations. The widely held suggestion, that there is a refractory period of about half a second following the making of a response, seems to have come from Telford (1931). It has been clearly refuted by Vince's experiments and by all the evidence since. Fitts (1951, p. 1323) suggested that there might be a refractory period of the same kind but with a duration of only about 100 millisec., and Davis (1957) has postulated a refractory period of about the same length following central activity rather than the actual making of a response.

B. Broadbent (1958, p. 280) has suggested that there may be a kind of quantising of perception into samples about a third of a second long. He further suggests that the subject can begin a new sample when S1 arrives and that delays to S2 are due to the data having to wait until the next sample before they can be dealt with. This theory, although fundamentally different in conception from the previous type has some similar consequences. It would, at first sight, account well for the data of the present experiment: the delays in the ungrouped cases when S2 arrived during TR1 were about what would be expected, and delays when S2 came during M1 would be due to S2 "missing" the next sample and having to wait for a third. Broadbent's theory would further offer an attractive explanation of the variability of delay when S2 arrived close to the beginning of M1 as this would also be about the end of the first sample after S1, so that in some cases S2 would be in time for the second sample but in other cases would have to wait for the third. The theory would not, however, account for the tendency to lengthening of M1 in cases in which *I'* was short. Further, one would expect that in cases in which S2 came during M1, TR1 and TR2 would have been positively correlated since short TR2 would be secured if TR1 ended and S2 arrived before the end of the first sample. Plotting TR2 against TR1 it was clear that the correlation if any, was negligible.

Both these two types of theory postulate that delays are caused by some process occupying a fixed time interval which is, as the theories are stated, independent of the length of TR1 and TM1, and would presumably not change with the amount of information conveyed by S1 or by the monitoring of M1. Both theories thus have the severe disadvantage that they require an *ad hoc* quantity to be postulated for the refractory period or quantised interval. If all the existing evidence is to be accounted for, such a quantity would have to be assumed to vary with circumstances in some way not as yet understood. The theories can thus, at the present stage at least, have only *post hoc* descriptive value and are not yet reliably predictive. They cannot, however, be refuted on present evidence, they can only be shown to be unnecessary. The crucial experiments have not yet been done. It would be instructive, for example, to try experiments in which the average reaction times would be longer than those obtained so far—for example by requiring multi-choice reactions. If under these conditions the delays were found to be no longer than those already observed, it would be evidence in favour of a theory such as that of Davis or Broadbent.

C. A different type of theory again states that the delays to S2 are due to what we have called the "Mowrer effect." Mowrer (1940) found that in a serial reaction time task signals arriving before (or after) a modal or mean interval were reacted to more slowly, and he "explained" the effect by saying that the subject's "expectancy" of a signal was then lower. This type of theory was originally suggested as a possibility by Hick (1948), and was espoused by Poulton (1950) and by Elithorn and Lawrence (1955). If specification of the actual amount of delay is not attempted the theory can fit a great deal of the evidence very well. Preference for other theories depends upon their giving a better *quantitative* fit to the data. Its lack of quantitative precision is indeed its main disadvantage and makes it again non-predictive. If all the data already accumulated were to be accounted for, the delays would have to vary with circumstances according to principles as yet unknown. Further, there is no mechanism in terms of which the effect can, as yet, be confidently understood, so that the theory is at present purely descriptive: the effect of "expectation" can only be observed *post hoc* from empirical data. Broadbent (1958, p. 272) refers to this theory as "parsimonious." It is so only if quantitative considerations are neglected. If they are taken into account, the additional postulates necessary to

explain variations in the amount of the effect make the theory very much less parsimonious than it appears at first sight.

D. Elithorn and Lawrence (1955) seem to imply the suggestion that the results of experiments using pairs of responses made by different hands could be accounted for in terms of cortical or other central interaction. It is reasonable to suppose that this might be substantial between bilaterally symmetrical effectors. It is true that Fraisse (1957) has shown that a signal may cause delay even if no overt response to it is required, but inhibition of action might well have significant effects in this respect. The complementary experiment in which responses to different signals would be made by widely separated effectors, e.g. jaw and foot, has not, so far as the writer is aware, been done.

E. Lastly, we may consider the hypothesis that there is in the central mechanisms a single-channel of limited capacity. This was implied in Craik's (1948) treatment, was made explicit by Hick (1948) and was developed by Hick and Bates (1950), the present author (Welford, 1952), Davis (1956, 1957), Fraisse (1957) and Broadbent (1957, 1958). In its bare essentials this theory assumes, firstly, a number of sensory input mechanisms each capable of receiving data and storing it for a limited period so that, for example, a short *series* of signals can be received as a unit. Secondly, it assumes a number of effector mechanisms containing both central and peripheral elements and capable of carrying out a series of actions such as the pressing and release of a key or a series of taps (Vince, 1949) as a single unit. Thirdly, between these two it postulates a single-channel decision mechanism. This is regarded as being of limited capacity in the sense that it takes a finite time to process information and can thus only deal with a limited amount of information in a given time.

It is further implied that sensory input data can be accumulated while the decision channel is occupied by dealing with previous data, and can be passed together to the decision channel as soon as it is free. Similarly the decision channel can "issue orders" to the effector side for a *series* of responses the execution of which can overlap with the decision channel's dealing with fresh input. Sensory feedback data from responding actions may, however, "capture" the decision channel, i.e. responses may be monitored.

This theory does not have the disadvantages of the others although it is not without its own difficulties, of which the most pertinent are perhaps three:—

(i) Some additional postulate is needed to account for "grouping." This, however, is a problem for any theory except, perhaps, that of Broadbent who might postulate that the subject would occasionally fail to begin a new sample with the arrival of S1, and that S2 would sometimes appear before he did so. A possible line of approach to the problem of grouping is to link it with the need to collect data over a period of time in order to distinguish a signal from "neural noise" (see e.g. Gregory, 1955), or with the fact noted in several studies (e.g. Piéron, 1923, Cheatham, 1952) that perception of a stimulus can in certain circumstances be modified or prevented by another stimulus coming a short time after.

(ii) The question arises of why movements are sometimes monitored and sometimes not. The present writer suggested (Welford, 1952) and Davis has reiterated (1956) that monitoring may be unnecessary when the accuracy required of the responding action is sufficiently low for it to be made ballistically without there being any appreciable likelihood that it will fail to be effective. If highly accurate movements are required, however, monitoring may be inevitable, and even when not strictly necessary it may

give the subject confidence. It is understandable that monitoring for either of these reasons would tend to drop out with practice, and we may note in support of this view that Davis's original subjects (Davis, 1956) and Marill's (1957) subjects, who are the two groups failing to show delays when S2 came after the end of TR1, were substantially more practised than others. Experiments comparing delays at various stages of practice could settle whether this explanation is correct.

(iii) The theory fits the evidence very reasonably well if we take the time for which the single channel is assumed to be occupied as equal to the total TR1. Such a formulation neglects, as Davis (1957) has pointed out, the fact that appreciable times are required for data to reach the cortex from sense organs and for efferent nerve impulses and muscular action to make a response effective. The theory would certainly not work if these times were deducted from TR1, as at first sight it would seem they should be, in order to arrive at the supposed time required for the decision mechanism to act in response to S1.

Any suggestion to overcome this difficulty must at present be speculative, but we may note that the difficulty would cease to exist if it could be shown that some minimum feedback from the responding action, indicating that it had begun, was necessary for the decision mechanism to be "cleared." If this were so the time taken to initiate a movement would automatically be included in the decision time, and there would be added a new time component of a few milliseconds for the feed-back signals to get back from the responding member to the brain. If this time was approximately the same as that required for a stimulus to reach the brain from an exteroceptor it would mean that reaction time would be a reasonable measure of decision time although the equating of the two would be in a sense fortuitous. An indication in favour of such a scheme is contained in the finding by Fraisse (1957) that delays following an S1 to which no response had to be made, and for which presumably the feedback did not occur, were shorter than when S1 was followed by an overt responding movement.

Future Research

We may end by briefly urging two general points for future studies which emerge from what has been done so far. Firstly a wide variety of theories will fit substantial areas of the facts, and distinction between the various theories can often only be made following a detailed *quantitative* analysis of the data. Any new theory should be accompanied by a review in quantitative terms of *all* the rather formidable mass of data so far collected.

Secondly, there seems to be a danger that the study of reactions to discrete pairs of signals will become unduly preoccupied with minutiae. There is a fairly obvious series of experiments still to be done with this technique, and doubtless if they were done they would suggest others. The kind of question these could settle would, however, almost certainly be answered incidentally, and a great deal of valuable information obtained besides, if attention was returned to the original stimulus to such studies, namely the analysis of continuous performance.

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APPENDIX A — Table of Means

All times in millisec.

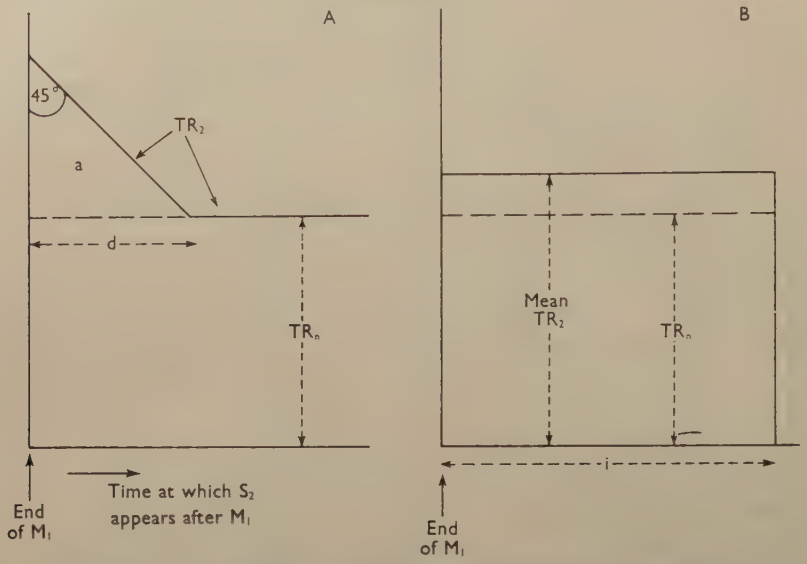
	Right after left or left after right		*Left after left*	
	Mean time	*Number of readings*	*Mean time*	*Number of readings*
Cases in which S2 came during TR1				
Ungrouped: *I*	186	61	224	20
TR1	317		306	
TR2	458		469	
Calculated TD2, i.e. TR2 + *I* − TR1	327		387	
Grouped: *I*	64	22	190	1
TR1	362		350	
TR2	433		310	
Reversed: i.e. M to S2 before M to S1				
I	56	16	—	—
TR1	552		—	
TR2	309		—	
Time from S1 to first M given, i.e. TR2 + *I*	365		—	
Cases in which S2 came during M1				
Ungrouped: *I′*	91	53	93	12
TM1	165		157	
TR2	393		409	
Calculated TD2 (assuming TF*b*1 = TM1), i.e. TR2 + *I′* − TM1	319		345	
Grouped: *I′*	32	13	—	0
TM1	305		—	
TR2	347		—	
TR2 in cases where S2 came after the end of M1, tabulated by interval between end of M1 and arrival of S2				
0–240	344	84	396	37
250–490	303	83	334	28
500–990	289	170	320	34
1,000–1,490	288	147	328	24
1,500–1,990	277	131	325	21
2,000–2,490	278	125	392	13
2,500–2,990	281	117	316	5
3,000–3,490	272	74	270	1
3,500 and over	291	33	—	—
			Right after right	
3,500 and over			298	393

APPENDIX B — Calculation of TF*e*

If there is delay due to TF*e*, reaction times to signals following closely after the end of M1 will be lengthened by an amount equal to the length of the delay (d) less the time (i) after the end of M1 at which S2 appears. The lengthening will equal d with $i = 0$ and be zero when $i \geqslant d$. For intermediate values of i, the net delay i.e. $(d - i)$ will have

a slope of $-45°$ as shown in Figure 5A. The *total* amount of delay is indicated by the 45° triangle with area a. What is required is to calculate d. Let us take first the simple case in which the frequency of signals at all intervals after the end of M1 is equal and d has zero variance. If we take an interval i ($\geqslant d$) the area a will be represented in the

FIGURE 5



A. Idealized diagram showing effects upon TR2 of delay due to monitoring the end of M1.

B. The same effect averaged over a period.

readings falling in this interval as a rise in the mean observed TR2, above a "normal" reaction time, as shown in Figure 5B. In this simple case the quantity d can be calculated from the formula

$$d^2 = 2a$$

taking a as the difference between the observed mean TR2 and the mean "normal" reaction time (TRn), multiplied by the interval i, i.e.

$$a = i\,(\overline{\mathrm{TR2}} - \overline{\mathrm{TR}n})$$

If d is subject to variation this formula requires modification. Writing $\bar{a}$ for the mean a, we have

$$2\bar{a} = \frac{\Sigma(d^2)}{N} = \bar{d}^2 + \sigma_d^2$$

so that

$$\bar{d}^2 = 2\bar{a} - \sigma_d^2$$

In the present experiment if we take $i = 500$ millisec. after the end of M1 we have mean TR2 = 323 millisec. Taking $\overline{\mathrm{TR}n}$ as 282 millisec. (the distribution of these times was shown in Figure 2) we have

$$\bar{a} = 500\,(323 - 282)$$
$$= 20500$$

If d were assumed to have no variance $\bar{d}$ would be the square root of twice this quantity, i.e. 202 millisec. Almost certainly, however, d will have some variance. An extreme case would be that in which $\sigma_d = \bar{d}$. In this case $\bar{d} = \sqrt{\bar{a}} = 143$ millisec. A reasonable *minimum* value for σ_d might be taken as that which would give the same coefficient of variation to d as to TRn. If we write $f =$ the reciprocal of the coefficient of variation, we have $\sigma_d = f\bar{d}$ and thus

$$\bar{d}^2 = 2\bar{a} - f^2\bar{d}^2 = \frac{2\bar{a}}{1 + f^2}$$

The value of f for the distribution of "normal" reaction times was found to be 0·1979 which yielded a calculated $\bar{d}$ of 199 millisec.

THE ROLE OF "ATTENTION" IN THE PSYCHOLOGICAL REFRACTORY PERIOD

BY

R. DAVIS

From the Institute of Experimental Psychology, University of Oxford

A set of experiments is described in which two consecutive signals, separated by a variable time interval, were presented to the subjects. Manual reaction times to these signals were recorded and the delays in the reactions to the second of the two signals were compared in situations where the subject was required to respond to both signals and where he was not required to respond to the first signal.

The magnitude of the delays found at any particular interval were the same in both situations, and there was not much difference between cases where the two signals were in the same sense modality (visual) or in different modalities (visual, followed by auditory). These results confirm the suggestion of the writer in a previous paper that there may be a common analysing and classifying system for auditory and visual information and also illustrate a new point. It is paying attention to a signal rather than performing any overt response to it which gives rise to delays in subsequent responses.

Introduction

In previous papers the present writer has suggested a model for the human operator responding to signals from the environment as a single channel through which one signal had to be cleared before another could be dealt with, in the manner suggested by Craik (1947 and 1948). The evidence was provided from experiments on reaction times to consecutive signals separated by short intervals of the type reviewed by Hick (1948) and Welford (1952). It was shown (Davis, 1956) that if a second signal was given 0·2 sec. or less after the first signal, a lengthened second reaction time to the second signal was obtained. In this case both signals were visual. In a further experiment (Davis, 1957) using an auditory signal followed by a visual signal, it was found that the magnitude of delays in the reaction time to the second signal was the same as when both signals were in the same, the visual, modality. It was therefore argued that there was some sort of functional convergence of signals arriving from different sense modalities; that the classifying of a signal and the organizing of a response to it was performed by the same central system, no matter by what sense modality the information arrived.

It might, however, be suggested that the common factor in all these experiments was that similar sorts of response had to be organized to the consecutive signals within a very short interval. The suggestion that interference on the response side is responsible for the delays is made unlikely by several previous observations. Thus the possibility of delays arising because of the limb not being in a position to make the second response, as a result of having performed the first, had been eliminated by using different hands for performing the consecutive responses. The fact that using two different hands would also involve the motor cortex in opposite hemispheres does not necessarily preclude the possibility of interference at this level, although it was shown (Davis, 1956) that the same order of delays is found whether different fingers of the same hand or of different hands were used, so that the effect seems identical whether the same or different halves of the motor cortex are involved in consecutive responses. Although Poffenberger (1912) reported a reduction in reaction times to single visual stimuli when using an

"uncrossed" relationship between the side of the visual field on which the stimulus was presented and the limb making the response, as compared with a "crossed" relationship, the size of the difference between the two situations was only of the order of 6 millisec. so that it would hardly be detectable in the present writer's experiments. Smith (1947) has cast further doubts on the likelihood of differential effects arising as a result of having sensory and motor areas for the reaction in the same or different hemispheres by showing that severing the inter-cortical neurones in man, while leading to a general lengthening of all reaction times, does not produce any more pronounced effect for the crossed sensori-motor reaction than for the uncrossed.

These observations suggest that the source of delays does not lie in the motor areas immediately concerned with the production of the response, or in any of the subsequent neural pathways. However, this evidence could hardly be regarded as conclusive and the writer therefore attempted to devise some behavioural means for deciding the probable locus of the delays. An approach was suggested by an observation of Drazin (unpublished), who noticed that delays in reaction times to a visual signal given shortly after a visual warning signal were nearly as large as would be expected if the subject had responded to the warning signal.

A series of experiments was therefore planned using both same-modality and cross-modality stimulation to compare the magnitude of the delays arising when the subject was required to respond to both signals, with the delays when the subject was required to respond to the second only of two signals. Whereas from Drazin's observations one might expect to find delays in responding to only the second when both signals are visual, it is by no means obvious what should happen when the two signals are in different modalities. The present experiments therefore involve the comparison of reaction times to the second of two signals separated by a variable interval under the following conditions.

Experiment 1. Visual-visual, responding to both signals.

Experiment 2. Visual-visual, responding to the second signal only.

Experiment 3. Visual-auditory, responding to both signals.

Experiment 4. Visual-auditory, responding to the second signal only.

Although the original experiment (Davis, 1956) used a visual-visual sequence, it was carried out using a different apparatus, for which the control simple reaction times were appreciably longer than in the present series. It was therefore necessary to repeat this condition with the present apparatus to obtain reliable comparisons.

Design

The general experimental design was the same as in previously reported experiments. The subject sat looking into a cardboard tube about 7 in. diameter and 3 ft. long, at the other end of which were mounted two neon bulbs, the centres of which were at eye level and spaced 2 in. apart. In all the experiments the subject wore padded earphones which served both to provide the auditory signal and exclude extraneous distracting noises. The signals to which the subject had to respond were either a 40 millisec. flash of a neon bulb or a click of moderate intensity in the earphones. The first visual signal in any pair was given on the left bulb, the second on the right. In Experiments 1 and 3, the subject responded to the first signal by pressing the left-hand reaction key and to the second signal by pressing the right-hand key. In Experiments 2 and 4 the single response to the second signal was made by pressing the right key. Reaction times were recorded by two sets of Dekatron counters in the manner previously described (Davis, 1957).

Procedure

On each day the subject had first to make 15 or so "simple reaction time" responses to a visual signal in Experiments 1 and 2 and an auditory signal in Experiments 3 and 4,

and the reaction times to the last 10 responses in each set were used as a control series. In all cases for the control series the single signal was presented at an interval after a warning, and this interval was varied randomly over the same range as the warning interval before the first signal in the test series, i.e. from 1·5 to 2·5 sec. in 0·1 sec. steps.

The experimental series was then presented. This consisted of 20 pairs of signals. The experimenter would say "Are you ready—now" and press the switch to start the timing apparatus. At a randomly varied interval between 1·5 and 2·5 sec. afterwards, the first signal would arrive and it would be followed at a randomly varied interval between 50 and 500 millisec. by the second signal. The range between 50 and 500 millisec. was divided into values differing by 50 millisec. and by drawing these values from a hat, a random arrangement in which each interval occurred twice in each series of 20 was ensured.

If for any reason a subject failed to make a response to any pair of signals, this interval was repeated later in the series. This happened rarely and the number of such occasion is shown in the results. Each subject was given several days' practice in the experimental conditions before any results were used. A further 20 experimental sessions were then given and the results of these are presented.

TABLE I

EXPERIMENTS 1 AND 2 (VISUAL-VISUAL SEQUENCE)

Means and Standard deviations of reaction times of intervals from 50 to 500 millisec. N = 40 in every case

	Experiment 1 *Visual-visual sequence, responding to both signals*				*Experiment 2* *Visual-visual sequence, responding to second signal only*	
	RT_1		RT_2		RT_2	
Interval (millisec.)	*Mean*	*S.D.*	*Mean*	*S.D.*	*Mean*	*S.D.*
Subject C						
50	159	14	286	27	254	38
100	153	15	224	34	220	33
150	162	16	204	21	190	24
200	168	26	185	26	176	23
250	164	24	172	25	163	20
300	157	17	166	15	163	22
350	163	21	158	12	164	25
400	155	15	149	17	159	18
450	163	21	152	18	160	18
500	160	18	153	13	152	17
Subject D						
50	173	19	230	28	244	48
100	171	16	211	21	207	41
150	171	18	202	18	181	25
200	177	20	187	15	167	13
250	169	18	173	18	161	12
300	167	14	157	10	161	13
350	171	21	153	15	154	22
400	175	19	150	15	164	12
450	173	21	145	10	150	14
500	178	19	140	10	155	10

Total number of readings requiring repeat for any reason:

	Experiment 1	*Experiment 2*
Subject C..	15 = 3·8 per cent.	16 = 4 per cent.
Subject D	24 = 6 per cent.	18 = 4·5 per cent.

Results

In Table I are shown the mean reaction times to the signals presented in Experiments 1 and 2. In Table II are shown the mean reaction times to the signals presented in Experiments 3 and 4.

TABLE II

Experiments 3 and 4 (Visual-Auditory Sequence)

Means and Standard deviations of reaction times for intervals from 50 to 500 millisec. N = 40 in every case

Interval (millisec.)	*Experiment 3 Visual-auditory sequence, responding to both signals*				*Experiment 4 Visual-auditory sequence responding to second signal only*	
	RT_1		RT_2		RT_2	
	Mean	*S.D.*	*Mean*	*S.D.*	*Mean*	*S.D.*
Subject C						
50	152	21	258	34	207	39
100	153	20	226	41	181	37
150	158	20	203	40	161	46
200	158	19	158	32	148	34
250	157	14	134	18	128	31
300	153	20	130	20	130	26
350	159	14	129	20	120	16
400	153	15	115	10	117	15
450	151	14	115	17	122	14
500	150	18	150	14	118	15
Subject D						
50	163	16	257	33	218	36
100	169	15	226	45	183	26
150	172	22	216	47	170	37
200	173	17	186	38	162	25
250	176	22	160	33	142	18
300	178	20	142	27	122	13
350	172	20	143	30	116	9
400	179	20	137	22	116	9
450	173	15	129	16	117	20
500	173	15	121	20	119	21

Total number of readings requiring repeat for any reason:

	Experiment 3	*Experiment 4*
Subject C	11 = 2·8 per cent.	17 = 4·3 per cent.
Subject D	25 = 6·3 per cent.	39 = 9·8 per cent.

In Figure 1*a* and *b* are plotted the mean reaction times to the second only of the two signals for all values of interval between them in Experiments 1 and 2. Figure 2*a* and *b* shows the corresponding values for Experiments 3 and 4. The control means shown are taken from the series of "simple reaction time" situations in which the subject was required to respond to the single occurrence of the appropriate stimulus (a visual signal for Experiments 1 and 2, an auditory signal for Experiments 3 and 4).

FIGURE 1a

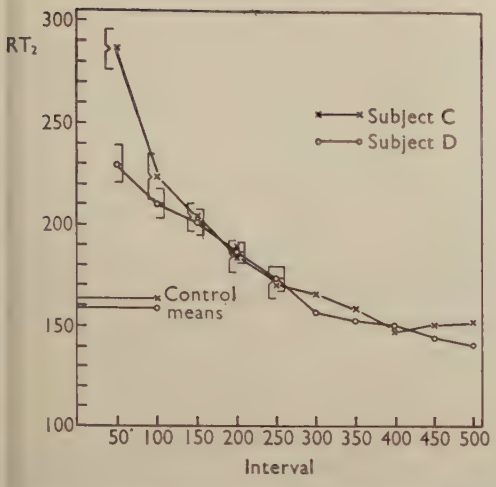
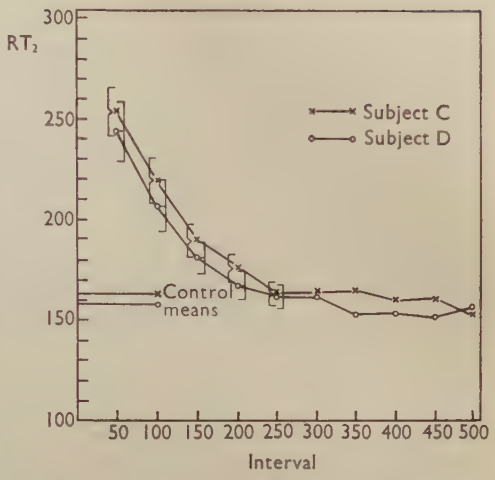


FIGURE 1b



Visual—visual sequence. Mean values of second reaction times for all values of interval. Confidence limits of two standard errors are marked on either side of the mean. The values of means for the control simple (visual) reaction times series are also shown.
1*a*. Experiment 1. Responding to both signals.
1*b*. Experiment 2. Responding to the second signal only.

FIGURE 2a

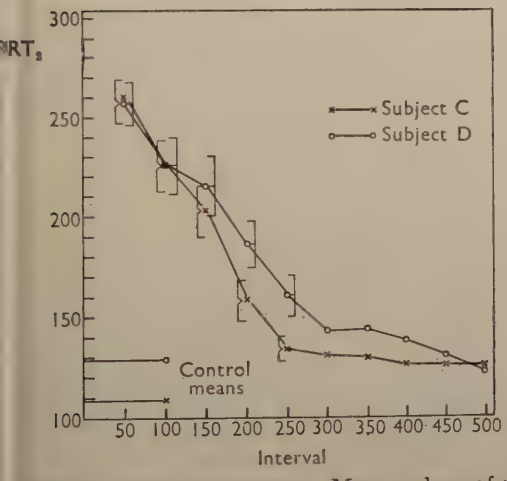
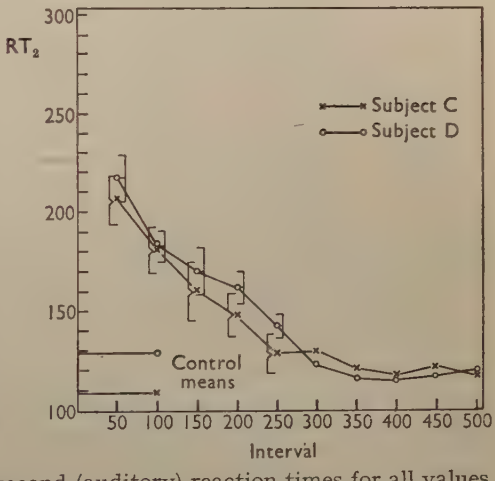


FIGURE 2b



Visual-auditory sequence. Mean values of second (auditory) reaction times for all values of interval. Confidence limits of two standard errors marked on either side of the mean. The values of means for the control simple (auditory) reaction times series are also shown.
2*a*. Experiment 3. Responding to both signals.
2*b*. Experiment 4. Responding to the second (auditory) signal only.

The following points may be noticed from these results.

(1) In all cases the delay in the response to the second of the two signals declines in a regular manner from a maximum at the shortest interval used (50 millisec.) and becomes negligible when the interval exceeds 250 millisec. This confirms the present writer's findings in previous experiments (Davis, 1956 and 1957) on the duration of the so-called "psychological refractory period."

(2) The delay disappears at this value of interval whether the second signal is in the same or different sense modality from the first (visual-visual or visual-auditory). This confirms the results of the experiment where the reverse sequence (auditory-visual) of signals was used (Davis, 1957) and supports the writer's suggestion that signals from visual and auditory modalities converge onto a common central channel for classification and analysis.

(3) A new fact is evident, that there is nearly as much delay present in the second response when the subject does not respond to the first signal as there is when he responds to both.

The differences in delays in the two situations are shown in Table III for the visual-visual sequence and in Table IV for the visual-auditory sequence. From Table III it can be seen that only a very small part of the delay was eliminated by not responding to the first signal when both the signals were in the same modality.

TABLE III

Comparison of the Delays in Reaction Time to the second of two Visual Signals under the Different Conditions of Making, or not Making, a Response to the First Signal

(Times in millisec.)

	1 *Responding to both (visual) signals (Experiment 1)*			2 *Responding to the second (visual) signal only (Experiment 2)*			*Difference in delays 1–2*
Interval (millisec.)	*Mean RT_2*	*Control*	*Delay*	*Mean RT_2*	*Control*	*Delay*	
Subject C							
50	286	163	123	254	163	91	32
100	224	,,	61	220	,,	57	4
150	204	,,	41	190	,,	27	14
200	185	,,	22	176	,,	13	9
250	172	,,	9	163	,,	0	9
Subject D							
50	230	159	71*	244	159	85	−14*
100	211	,,	52	207	,,	48	4
150	202	,,	43	181	,,	22	21
200	187	,,	28	167	,,	8	20
250	173	,,	14	161	,,	2	12

* This is the anomalous result of Subject D.

There is in this table clearly shown an anomalous result of subject D at the interval of 50 millisec. The reaction time to the second signal was actually shorter in responding to both signals than when responding only to the second. The writer is inclined to regard this as an anomaly peculiar to this subject in Experiment 1, as there is no trace of this abnormal shortening in any other of the experiments in which this, or any other subject, was used. This subject in Experiment 1 showed every sign of distress and annoyance if a rather long reaction time was recorded and tended to anticipate signals to a greater extent than the other subject. Thus 6 per cent. of the total number of pairs of stimuli given had to be repeated for subject D, most of these being necessary in order to repeat cases in which one or other response was anticipated. It might have been that the shorter second reaction times at 50 millisec. interval were the result of "grouping," that is, delaying the first response and treating the two signals as a complex, to which a complex, double, response is made (Hick, 1948; Vince, 1948 and 1950). If this were so, then we

should expect the first reaction times to be lengthened by at least the value of the interval between the two signals when compared with the reaction time to a single signal, i.e. by at least 50 millisec. for a 50 millisec. interval. Thus, if all or most of the responses were being grouped we should expect the mean reaction time for the first element of the grouped response to be at least $RT_N + 50$ millisec. (where RT_N = simple reaction time control). Although the mean RT_1 at the 50 millisec. interval for subject D is larger than the control value, the difference is only small, 173 — 159 millisec., and although this might indicate that the subject was waiting before performing the first response, it is clear that none of the advantages of "grouping" would be gained with such a short wait.

TABLE IV

COMPARISON OF THE DELAYS IN REACTION TIME TO AN AUDITORY SIGNAL PRECEDED BY A VISUAL SIGNAL UNDER THE DIFFERENT CONDITIONS OF MAKING OR NOT MAKING A RESPONSE TO THE VISUAL SIGNAL

(Times in millisecs.)

	1 *Responding to both (visual-auditory) signals (Experiment 3)*			2 *Responding to the second (auditory) signal only (Experiment 4)*			
Interval (millisec.)	*Mean RT_2*	*Control*	*Delay*	*Mean RT_2*	*Control*	*Delay*	*Difference in delays*
Subject C							
50	258	109	149	207	109	98	51
100	226	,,	117	181	,,	72	45
150	203	,,	94	161	,,	52	42
200	158	,,	49	148	,,	39	10
250	134	,,	25	128	,,	19	6
Subject D							
50	257	129	128	218	129	89	39
100	226	,,	97	183	,,	54	43
150	216	,,	87	170	,,	41	46
200	186	,,	57	162	,,	33	24
250	160	,,	31	142	,,	13	18

It is still possible that some responses may have been grouped and others performed as distinct reactions. Merely examining the mean RTs would not enable us to detect this possibility. Thus the slightly larger mean RT_1 of 173 millisec. might be composed of the first reaction times for both discrete and grouped responses. Examining the distributions of reaction times shows this to be unlikely, but a more precise test can be made in the following manner. For each interval there is a distribution of RT_1s and a distribution of RT_2s, both approximately normal (although slightly skewed towards the larger values). If these distributions result from a mixture of grouped and ungrouped responses, then this would mean that relatively long RT_1s would be associated with relatively short RT_2s (the "grouped" responses) whereas the shorter RT_1s would be associated with the longer RT_2s (the discrete responses). Thus the product moment correlation coefficient for RT_1 and RT_2 pairs taken over the whole distribution should be negative (although within each homogeneous group composing the distribution it may be positive).

In fact the coefficient for RT_1 and RT_2 pairs at this interval was found to be positive 0·77 ($p = 0{\cdot}05$), which suggests that the whole distribution forms a homogeneous or nearly homogeneous group. But it was established above that if we assumed we were dealing with a distribution in which all, or nearly all, responses were grouped, then the time the subject was waiting (about 15 millisec.) was so short that none of the advantages to be gained by grouping could be achieved. Therefore if the distributions form a homogeneous or nearly homogeneous group, as the positive correlation coefficient suggests, then this can only be because they are all, or nearly all, non-grouped responses.

It therefore seems that the anomalous result of subject D cannot be explained by the grouping hypothesis.

From Table IV it can be seen that although some of the delay is eliminated by not having to respond to the first of the two signals when they are in different modalities, the major part of it still remains.

Discussion

It might be argued that there should be a difference in the delays arising from same modality and cross modality stimulation when no response is required to the first signal. Thus in the visual-visual situation, even though consecutive signals were given on different neon bulbs, they were so close together and the signals were so similar that the subject may have found it difficult to identify the second signal as the one for which a response was required unless he had already noticed the first signal. That is, the subject may have had to pay attention to the first signal even though no response was required to it.

When different modalities are used for first and second signals there would seem to be no need for the subject to attend to the first signal. It might be possible for the subject to set himself for one kind of stimulation and neglect any stimulation arriving via other modalities, for this information is not necessary for him to be able to identify the signal to which he must respond. The results indicate, in these experiments, that the subjects were unable to perform in this sort of way. The occurrence of a visual signal caused almost as much delay in the response to an auditory signal given shortly afterwards as it did in the response to another visual signal given at the same time afterwards. We therefore need to explain why a signal which the subject could afford to neglect completely had such a disturbing effect on a subsequent response.

It should be noticed that the experimental conditions were such that both the visual and auditory stimuli were highly "compelling" from the subject's point of view. Practically the whole of his visual field was occupied by the black cardboard tube down which he was looking, and the only change in stimulation in the visual field was the flash of the neon bulb. He was also wearing padded earphones so that there was little auditory variation apart from the sound of the click. In these circumstances it is perhaps not surprising that a flash from the neon disturbed the response to the auditory signal. It has high "novelty" in Broadbent's sense (1953). In other circumstances where the flash might not be so compelling, it is likely that such a change could occur without the detrimental effect on the subsequent response. There seem in fact to be two distinct reasons why a previous signal might have a disturbing effect on the response to a subsequent signal. The first reason is that attention may be directed to the first signal as a result of its distracting effect, and this kind of effect might be expected only when the first stimulus was of a fairly compelling nature, as in the present series of experiments. The second reason is that some sort of discrimination of the first signal may require the direction of attention. The interfering effect of this discrimination on the response to a second signal would not depend on the compelling nature of the first signal. The results from the experiments where no first response was required to the first signal might therefore be interpreted in the following manner. The fact that considerable delays occurred in the visual-auditory situation suggests that the visual signal was compelling attention because of its distracting effect. The fact that delays were even longer in the visual-visual situation suggests that there may be a further effect due to the subject having to perform some sort of discrimination of the first signal.

In view of the experimental design, a further possibility must be considered. The subject performed Experiments 2 and 4 (the single response experiments) in parallel with Experiments 1 and 3 (the double response experiments) respectively. After

completing a single session under the conditions of Experiment 1, they would have a short rest and then do a session of Experiment 2, and a similar arrangement was used for Experiments 3 and 4. It could be argued that the reason for the delays in Experiments 2 and 4 was that the subjects were having to inhibit an overt response to the first of the two signals which they had learned to perform in Experiments 1 and 3. In order to provide some check on this possibility, the results for subject D were taken under two different conditions in Experiment 4. Half of the trials were done at the same sessions as the latter half of Experiment 3 and the other half were done some two months later. In the meantime the subject had had a complete break from this type of experiment. The results shown above for Experiment 4 in fact group these two sets together, but in Table V are shown the values of the auditory reaction times under the two different conditions.

TABLE V

MEANS AND S.D.S OF REACTION TIMES (IN MILLISEC.) IN RESPONDING TO ONLY THE SECOND OF TWO SIGNALS (VISUAL-AUDITORY)

(A) When the trials took place on the same days as those for Experiment 3.
(B) When the trials took place after 2 months' rest from this type of experiment.

Interval (millisec.)	*20 Trials in parallel with Experiment 3*		*20 trials 2 months later*	
	M	*S.D.*	*M*	*S.D.*
Subject D only				
50	234	32	202	29
100	201	20	166	18
150	191	33	148	47
200	180	19	146	17
250	149	17	130	19
300	122	14	123	11
350	114	12	119	9
400	113	11	119	5
450	119	27	116	7
500	113	13	125	25

The fact that there is an appreciable difference in the delays under the two conditions shows that there was an interfering effect of the two response situation on the single response situation when they were done in parallel. This effect could be ascribed either to the subject having to pay attention to the visual signal in order *not* to respond to it, or to the effect of his having to inhibit a response before it became overt. Further investigation would be needed to sort out these possibilities.

On the other hand, the single response sessions performed on their own, two months later, still show considerable delays at the two shortest intervals between signals. This tends to indicate that delays arise not as the result of any response produced interference (e.g. as in the proprioceptive feedback theory of Welford) but as a result of the perceptual demands made on the subject. The writer suggests that these delays will occur whenever a subject's attention is taken up, for whatever reason, whether or not an instrumental response to the attention-provoking signal is made. In terms of the single channel conception of the human operator, the channel becomes occupied (and hence refractory to other signals) by any compelling ("novel," intense) signal or by any signal which requires some form of discrimination. This division roughly corresponds to that between signals which are likely to have

general biological importance (Broadbent 1953) and signals which are important for the task in hand.

The present writer's results support those of Fraisse (1957), who used a very similar situation. Visual-visual, auditory-visual and visual-auditory sequences were used and in some experiments the subject was not required to respond to the first of the two signals. The interval between signals was randomly varied between values of 150, 300, 450, 600, 800 and 1200 millisec. Fraisse reaches similar conclusions, (1) that the reaction time to the second signal is prolonged whether the first signal is of the same or different nature, (2) that there is still a considerable delay when no response is required to the first signal.

On other issues Fraisse differs from the present writer. Thus he still finds small delays in the second reaction time when the interval between signals is 300 millisec., and suggests that Welford's feedback theory may be appropriate to explain them. However, these are small delays and he himself points out that his subjects had only two short practice sessions before results were recorded. He also supposes that the greater delay arising when the two signals are both visual might indicate that there is a peripheral interference effect due to the inertia of the visual receptors. This is unlikely, for the fact that the visual receptors can follow much higher rates of stimulation than those employed in any of the "psychological refractory phase" experiments would seem to exclude any purely peripheral source of delay, and the account given by the present writer provides an alternative explanation.

The main conclusion to be drawn from the series of experiments described in this paper is that most of the delay in responding to the second of two signals separated by a short interval is present whether the first signal is responded to or not. The delays arise as a result of paying attention to the first signal, rather than the performance of any overt response to it.

The writer wishes to thank Professor G. Humphrey and Professor R. C. Oldfield for the provision of experimental facilities at the Oxford University Institute of Experimental Psychology and Dr. H. Kay for his continued interest. The work is a continuation of that commenced under a grant from the Conditional Aid Fund by the M.R.C. and D.S.I.R. Joint Committee on Individual Efficiency in Industry.

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SOME FACTORS AFFECTING TASTE SENSITIVITY IN MAN. I:

Food Intake and Time of Day

BY

ROY YENSEN

*From the Psychological Laboratory, Cambridge**

In most studies involving taste stimuli it is assumed that the sensitivity of the taste mechanism is relatively stable. At the same time the results of some experiments with animals and man suggest that alterations in the "milieu interieur" may be accompanied by significant changes in taste sensitivity. Some of these reports are critically reviewed and the effects on taste sensitivity of certain changes in the general physiological state of the organism are described.

A highly reliable method of assessing taste sensitivity to the four basic taste qualities is developed. Taste sensitivity of human subjects is studied under conditions of normal food intake, during fasting and following variations in the caloric value of the lunch time meal. The effects on taste sensitivity of loss in total body salt and loss in total body water are also reported in the second and third papers respectively.

Depletion of total body salt was induced by forced sweating coupled with a very low salt intake during the treatment periods. Water was allowed *ad libitum*. The general symptoms experienced by the subjects, recorded losses in body weight, and analyses of venous blood samples showed that definite losses in body salt were incurred by the subjects during these periods.

Total body water was depleted by having the subjects refrain from any deliberate intake of water coupled with a diet of very low water content over treatment periods of up to 66 hr. Again the general symptoms experienced by the subjects, the recorded losses in body weight and the results of analyses of venous blood samples showed that definite losses in body water were incurred by the subjects during these periods.

In view of the need to control overall dietary intake and general routine over prolonged periods, it was found possible to carry out these experiments on only a small number of subjects. It is, however, felt that the lack of basic data in the problem under investigation and the need for intensive analyses, justifies a study on a smaller scale than is usual in psychophysical experiments.

The results indicate that in the fasting state slight fluctuations occur during the day in sensitivity to all four taste qualities with greatest sensitivity at the 11.30 a.m. session. Systematic fluctuations also occur under conditions of normal intake of food. In the hour or so following a normal meal sensitivity is significantly depressed and increases again after 3 to 4 hr. The degree of decrease following a normal meal appears to be related to the caloric value of the meal. The smaller the caloric value, the less is the decrease in sensitivity.

Loss of body salt is accompanied by a significant increase, and loss of body water by a significant decrease in sensitivity to salt. These changes appear to be of a specific character and do not effect the thresholds for taste qualities other than salt.

The probable function of these sensitivity changes in relation to the homeostatic mechanisms of the body is considered.

It is concluded that certain changes in the physiological state of the organism do influence taste sensitivity and that, therefore, these variables (i.e. time of day, dietary intake, salt balance and water balance) must be controlled in studies involving taste.

In the course of a recent investigation it was necessary to establish whether such factors as dietary intake and time of day are variables influencing taste sensitivity in man.

* Now at the School of Applied Psychology, University of New South Wales.

Of the five relevant studies in the literature three (Goetzl *et al.*, 1950; Hammer, 1951; Irvin and Goetzl, 1952) suggest that these variables probably do influence taste sensitivity, whilst the other two (Janowitz and Grossman, 1949; Meyer, 1952) yielded negative results.

Goetzl and Stone (1947) report that diurnal variations in olfactory acuity do occur and that they may be related to the sensation complex of appetite and of satiety. In an endeavour to check on this finding and to extend it to taste, Janowitz and Grossman (1949) determined the thresholds for the recognition of the taste of salt and of sugar and the smell of coffee at 10.30 a.m., just prior to the noon meal and at 2 p.m. In another series of tests the effect of 10 mgm. of D-amphetamine on these taste and smell thresholds was determined. They found "only minor variations in the acuity of the sense of taste and olfaction during the day" (p. 222), and concluded that these variations are not directly related to the presence or absence of hunger sensations and appetite.

However, Goetzl *et al.* (1950), after determining the threshold for sucrose at 10 a.m., 11 a.m., 1.30 p.m. and 4.30 p.m., report the existence of significant diurnal variations in taste sensitivity. In general, the pattern of variation showed an increase in sensitivity to sucrose from 10 a.m. to 11 a.m. with a decrease at 1.30 p.m. (if a normal lunch was eaten) and an increase at 4.30 p.m. When lunch was omitted the decrease in sensitivity at 1.30 p.m. did not occur. On the basis of this pattern of variation and the subject's reports of hunger before the noon meal and of satiety following it, the authors suggest a relationship between the sense of taste for sucrose and the sensation complex of appetite and of satiety. In a similar study of diurnal variation in the taste threshold for sodium chloride, Irvin and Goetzl (1952) report the existence of a similar pattern of variation and reach the same conclusions.

After noting the apparent disagreement between the findings of Janowitz and Grossman (1949) and those of Goetzl *et al.* (1950), Hammer (1951) carried out experiments to check whether diurnal fluctuations occur in thresholds of odour (coffee), taste (hydrochloric acid), and vision (critical flicker frequency). Testing times of 8 a.m., 11 a.m., 1 p.m., 2 p.m., and 4 p.m. were used, with variations in conditions of normal, large, late, and no lunch. From his results Hammer concludes that "odor, taste, and flicker-fusion thresholds fluctuate in a diurnal pattern which is related to food intake. Diurnal variations in sensory thresholds are influenced by a number of physiological factors as well as the interactions among these factors. In this study the conditions were such that not only food intake, but also fatigue and general bodily condition were factors which affected the pattern of variation" (p. 411). The patterns of variation for the thresholds of taste and smell reported by Hammer are quite similar to those found by Goetzl *et al.* and Irvin and Goetzl.

Meyer (1952) carried out successive determinations of sweet, salt and bitter thresholds at 3-hourly intervals during a 34-hr. fast by 9 subjects. He reports that "no systematic changes were found for any of the three modalities" (p. 376).

Evaluation of these findings is rather difficult because of certain defects in the experimental procedures used by the various investigators. Space does not allow a detailed criticism of each of these studies, but several of their unsatisfactory aspects will be noted.

For example, so far as their reports indicate, Janowitz and Grossman, Goetzl *et al.* and Meyer did not give their subjects any practice before critical testing was begun. Nor do any of these authors or Hammer satisfactorily demonstrate that their threshold measures are reliable. In each study the conclusions are based on only one, or in Meyer's study two, threshold determinations per session. Goetzl *et al.*, Irvin and Goetzl and Hamer did, however, repeat sessions over a number of days,

thus increasing the total number of threshold determinations for a given condition. The overall dietary intake and the general routine of the subjects was not fully controlled in any of these studies. Nor were the methods used to establish taste thresholds very satisfactory. Janowitz and Grossman and Hammer used a method based on Richter and MacLean's (1939) "choice method no. 2." In this method successive "tastes" of the stimuli are allowed without any rinsing and drying of the tongue and without any time lag between "tastes" to allow for recovery of the taste receptors. Such a method may well yield confusing results since a given "taste" of the test solution may be contaminated as a result of the state of the taste receptors due to the immediately preceding "taste." Goetzl *et al.*, Irvin and Goetzl, and Meyer, used a drop method of stimulation, but do not report any special precautions taken to ensure that the same region and area of the tongue were stimulated throughout testing. The great difficulty with the drop method is to ensure that the solution falls on the same spot and spreads over the same area each time the stimulus is delivered, for variations in the region and in the area stimulated result in variation in the threshold (Bujas and Ostojcic, 1941).

The present communication reports observations on systematic fluctuations in taste sensitivity during the course of the day under varying conditions of dietary intake. More specifically answers were sought to the following questions:—

(1) Do systematic fluctuations occur in taste sensitivity during the course of a normal day?

(2) Are these fluctuations related to the intake of food?

In this study taste sensitivity is assessed in terms of the absolute thresholds for taste of the accepted four basic taste qualities of sweet, salt, sour and bitter (Geldard, 1953; Moncrieff, 1951; Pfaffmann, 1951).

In view of the lack of conclusive evidence as to what variables are relevant in studies of taste sensitivity, it seemed preferable, at this stage, to carry out intensive work on a small number of subjects over whom fairly rigid control of general routine, overall dietary intake and general well-being could be exercised for prolonged periods.

Method

The stimuli consisted of 8 to 10 ml. of distilled water solutions of sucrose, sodium chloride, sulphuric acid, and caffeine for the sweet, salt, sour, and bitter test solutions respectively. The temperature of all solutions was maintained within 20° to 21·5° C. during testing. For each taste quality a series of solutions was mixed which ranged, for a given subject, from well below to well above his threshold level under each condition of testing. The series were graded in steps of concentration (grams of solute per 100 c.c. of solution) of 0·05 of a log unit for all test solutions.

The subject was instructed to respond in terms of the presence or absence of the specific taste quality under consideration. He was also encouraged to report on any other aspects of the stimuli and of the test in general which he felt might be relevant.

During testing the subject was seated comfortably at a table with a large enamelled funnel situated at his side or just in front of him, whichever he preferred. All used rinse water and solution were expectorated into this funnel which drained through tubing to a container under the table. An 8 oz. glass and ample supplies of rinse water were on the table. Across the centre of the table a large screen was erected which shielded the tester and his array of test solutions from the subject's view. The stimuli were passed around this screen to the subject who tasted them (see below), and replaced the glass on the table in front of him, from where it was retrieved by the tester. In this way the subject was kept in complete ignorance of the strength or number of solutions being used, and of the nature of the record being kept by the tester. At no time, even during repeated testing, was the subject told of the level of his threshold until all testing was completed.

Each stimulus was presented individually in an 1½ oz. medicine glass. The subject sipped the entire contents of the glass and held it on his tongue, until he perceived the

specific taste quality under consideration or until 4 sec. had elapsed, whichever occurred sooner. He then expectorated the fluid, rinsed his mouth with distilled water and reported his judgment. Immediately prior to each trial the subject rinsed his mouth thoroughly with distilled water and dried it by pressing his tongue against the roof of his mouth and swallowing. All rinse water and test solutions were expectorated. Stimuli were presented in an ascending order of concentration with a rest pause of 30 sec. between the rinse following the previous trial and the rinse before the next trial. Thus the rate of presentation of stimuli was approximately one per minute.

The procedure, then, was for the subject to "rinse," "dry his mouth," "taste," "rinse," "report," "rest," "rinse," and so on, as each stimulus was presented, until two successive positive reports were made. In this manner four series of trials were administered with the starting point of each run varied. At the discretion of the tester some runs were terminated at the first positive report, but this only when the record of the subject indicated that the level of the first positive report was highly consistent with previous reports. Rest pauses of 3 min. were given between runs. The threshold was taken as lying halfway between the lowest of the two successive stimuli to which positive responses were made, and the concentration immediately below it. Each session lasted 30 to 35 min.

No eating, drinking or smoking was allowed within at least ½ hr. of any testing session. Testing was cancelled if the subject did not feel well. Checks were made as to the presence or absence of coating on the subject's tongue and a record of this was duly made at the beginning of each session. (Slight coating was occasionally recorded. Since this did not appear to have any influence on sensitivity, it is not discussed in presentation of results.) Prior to each session the subject rinsed his mouth very thoroughly with distilled water. During prolonged testing periods the subject carried on with his normal duties, but kept regular hours of sleep, meal times, etc., and avoided any excessive or unusual physical exertions. Unless the experimental purpose dictated otherwise, the overall caloric value of the subject's daily dietary intake was controlled at a level estimated to be approximately equal to his usual dietary intake.

Before any critical testing was begun the subject was given practice sessions, until his threshold values for a specific taste quality became relatively stable under given conditions of testing. Once such stability in the level of the subject's threshold was attained, critical testing under the experimental conditions was carried out without a break until the particular experiment was completed.

Reliability was checked by calculation of the standard errors of the means of threshold measurements taken during the various experiments.

Experiment I

Procedure

Thresholds for sweet, sour, bitter, and salt were determined at 9 a.m., 11.30 a.m., 2.15 p.m. and 4.45 p.m. for 4 days each and for sweet, sour, and bitter for a further 2 days each at 11.30 a.m., 2.15 p.m. and 4.45 p.m. under conditions of normal intake of food. Two subjects were used. To standardize the conditions of testing from day to day, the general routine and dietary intakes of the subjects were controlled over all test days. Regular hours of sleep (retire at 10.30 to 11 p.m., rise at 7.30 a.m.) and meal times (breakfast 7·45 a.m., morning coffee 10·15 a.m., lunch 12·30 p.m., afternoon coffee 3.15 p.m. and dinner 7 p.m.) were kept by both subjects. They carried out their usual duties between test sessions (subject H: typist; subject R: research student). The caloric value of the daily dietary intake from all sources for subject H was maintained at approximately 2,100 calories (consisting of about 36 per cent. carbohydrate, 12 per cent. protein, 52 per cent. fat), and for subject R at approximately 2,650 calories (consisting of about 46 per cent. carbohydrate, 11 per cent. protein and 43 per cent. fat). The approximate caloric intake at each meal is shown in Table I. Water was allowed *ad libitum*.

Results

The mean threshold values for sweet, bitter, sour and salt at each time of day are presented in Figures 1*a* and 2*a* for subjects H and R respectively. By comparing the mean values of thresholds for sweet, bitter and sour determined at 9 a.m., 11.30 a.m., 2.15 p.m. and 4.45 p.m. (curve ——) with those gained at 11.30 a.m., 2.15 p.m. and 4.45 p.m. (curve ....), it can be seen that the existing variations are not due to the effects of previous testing on the same day.

The results for each subject on each taste quality were analysed separately by Analysis of Variance (Guilford, 1950) and in all cases a significant variation ($P < 0.01$) between times of day was found.

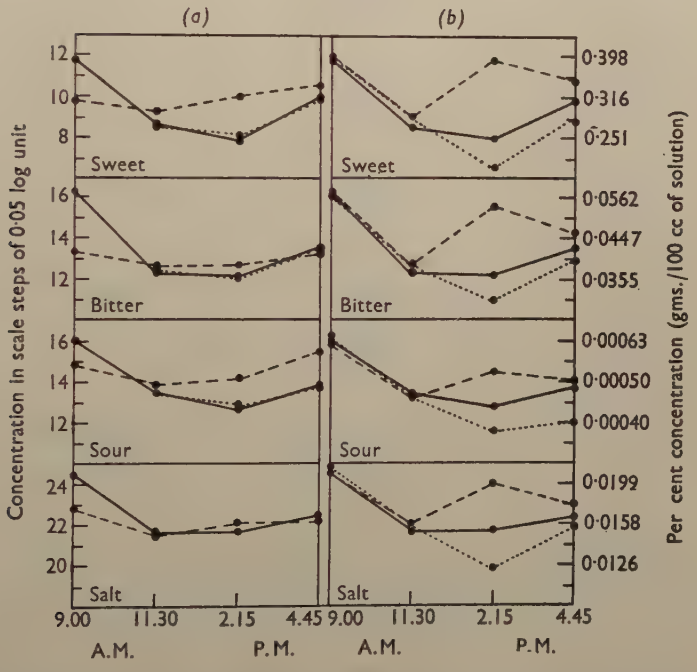
TABLE I

CALORIC VALUES OF MEALS DURING "NORMAL" TEST DAYS. SUBJECTS H AND R

(Calculated from McCance and Widdowson, 1946)

Meal	*Caloric Value*	
	H	*R*
Breakfast	510	760
Morning coffee	85	125
Lunch	660	810
Afternoon coffee	85	125
Dinner	760	830
Total	2,100	2,650

FIGURE 1

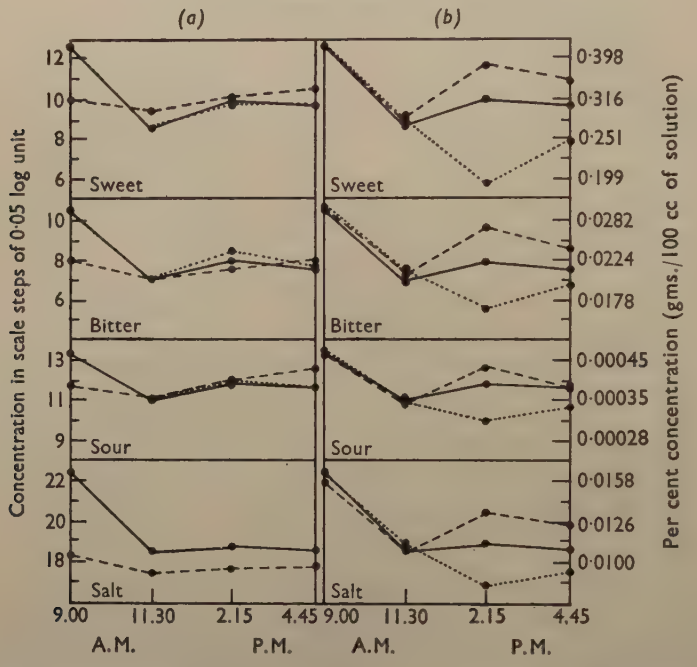


Mean taste thresholds for subject H. (*a*) "normal" and "fasting" test days (——— normal, four sessions/day; ..... normal, three sessions/day; - - - - fasting); (*b*) "normal", "large lunch" and "no lunch" test days (——— normal; - - - - large lunch; ..... no lunch).

The concentration corresponding to scale step 0 for salt = log $\bar{3}\cdot10$ = 0·0013 per cent.; for sweet = log $\bar{1}\cdot00$ = 0·1000 per cent.; for sour = log $\bar{4}\cdot00$ = 0·00010 per cent.; for bitter (caffeine) = log $\bar{3}\cdot95$ = 0·0089 per cent.

The patterns of variation during the day are similar for all taste qualities with both subjects in that all the 9 a.m. threshold values are significantly larger than either the 11.30 a.m., 2.15 p.m. or the 4.45 p.m. values (t-tests; $P < 0.01$). and in all cases the 4.45 p.m. values fall between the 9 a.m. and the 11.30 a.m. values (except for the salt 4.45 p.m. value of subject R which is the same as his 11.30 a.m. value). There is, however, one difference. The 2.15 p.m. values on all qualities are larger than the 11.30 a.m. values for subject R, whereas for subject H this is not so. It was thought that this may be due to the difference in the size of lunch consumed by the two subjects. Subject H had about 660 calories as compared with 810 calories for subject R.

FIGURE 2



Mean taste thresholds for subject R. (*a*) "normal" and "fasting" test days (——— normal, four sessions/day; . . . . . normal, three sessions/day; - - - - - fasting); (*b*) "normal," "large lunch" and "no lunch" test days (——— normal; - - - - - large lunch; . . . . . no lunch).

The concentration corresponding to scale step 0 for salt = log $\bar{3}$·10 = 0·0013 per cent.; for sweet = log 1·00 = 0·1000 per cent.; for sour = log 4·00 = 0·00010 per cent.; for bitter (caffeine) = log $\bar{3}$·95 = 0·0089 per cent.

Experiment II

To check whether the size, or rather, the caloric value, of the lunch influences the level of sensitivity at the 2.15 p.m. session, tests were carried out for 2 days each on subjects H and R in which conditions, as above, were held constant, except for the caloric value of the lunch. This varied from "no lunch" for both subjects to a "large lunch" of about 1,060 calores for subject H and about 1,270 calories for subject R. To keep the overall daily caloric intake constant, the evening meals were increased after lunch was omitted, and decreased when a large lunch was consumed.

From Figures 1*b* and 2*b* it can be seen that the 2.15 p.m. level of sensitivity is influenced by the caloric value of the lunch time meal and that there is, in some cases, a carry over of this influence to the 4.45 p.m. session. Statistical analysis of these results reveals that for both subjects, over all taste qualities, the ingestion of a large lunch resulted in a significant increase ($P < 0{\cdot}01$) in the 2·15 p.m. threshold values, whereas the omission of lunch resulted in a significant decrease ($P < 0{\cdot}01$) in the 2.15 p.m. values as compared with the 11.30 a.m. values. For both subjects, on all taste qualities, the "normal lunch" 2.15 p.m. and 4.45 p.m. values lie between those of the "no lunch" and "large lunch" conditions (Figs. 1*b* and 2*b*).

It appears, then, that taste sensitivity after the lunch time meal is influenced by the caloric value of the food ingested by the subject at that meal. This fact probably accounts for the difference in the patterns of variation between subjects H and R over the 11.30 a.m. and 2.15 p.m. sessions in Experiment I, i.e. it is suggested that, if subject H had, under "normal lunch" conditions, eaten a somewhat larger lunch, then her 2.15 p.m. thresholds would, as was the case for subject R, have been larger than her 11.30 a.m. thresholds.

Experiment III

As a further check on the influence of food intake and to see whether taste sensitivity varies with the time of day, independent of food intake, thresholds were determined on subjects H and R in the fasting state at 9 a.m., 11.30 a.m., 2.15 p.m. and 4.45 p.m. Tests were made for 2 days on each of the four qualities with each subject. The test procedure was the same as that used in the earlier experiments. The subjects had their usual dinner on the evening preceding test days and then refrained from all food intake until after the 4.45 p.m. session (water was allowed *ad libitum*). The evening meal on the test days was as large as the subjects desired. Test days were separated by one day of normal food intake. The subjects reported feelings of increasing hunger and sometimes of lassitude and weakness towards the end of the fasting periods.

The mean results for each taste quality are shown in Figures 1*a* and 2*a*. It may be seen that these results differ considerably from those obtained under conditions of normal food intake. Two-way analysis of variance of the results from the "normal" and "fasting" days showed a significant interaction ($P < 0{\cdot}01$) for all taste qualities with both subjects. Thus, the patterns of variation in taste sensitivity under these two conditions are significantly different. The influence of food intake on sensitivity is further shown by the fact that for all qualities and for both subjects the 9 a.m. "normal diet" thresholds are significantly higher ($P < 0{\cdot}01$) than the 9 a.m. "fasting" thresholds.

Analysis of variance of the "fasting" data reveals a significant variation ($P < 0{\cdot}01$) with time of day for both subjects over all qualities, except bitter for subject H. Similarity in the patterns of variation between taste qualities and between subjects exists in that all show a slight decrease from the 9 a.m. thresholds to the 11.30 a.m. thresholds, which are, in all cases, the lowest values recorded throughout the day.

The highly reliable nature of the measurements made in these experiments is indicated by the relatively small standard errors shown in Table II.

Discussion

It appears, then, that under conditions of normal intake of food systematic fluctuations in taste sensitivity do occur during the day and that, to some extent, they are dependent on food intake. This finding agrees with the results of Goetzl

et al. (1950) for sweet, Hammer (1951) for sour and Irvin and Goetzl (1952) for salt, and is contrary to the results reported by Janowitz and Grossman (1949) and Meyer (1952).

TABLE II

MEANS AND STANDARD ERRORS OF SOME COMBINED DATA FROM "NORMAL," "LARGE LUNCH" AND "NO LUNCH" TEST DAYS. SUBJECTS H AND R

		H		*R*	
Time	*No. of determinations*	*Mean**	*S.E. (of mean)*	*Mean**	*S.E. (of mean)*
	Sweet				
9·00	32	11·87	0·097	12·59	0·094
11.30	40	8·75	0·092	8·75	0·092
	Bitter				
9.00	32	16·31	0·130	10·53	0·114
11.30	40	12·52	0·103	7·27	0·090
	Sour				
9.00	32	16·06	0·089	13·34	0·091
11.30	40	13·37	0·088	11·05	0·079
	Salt				
9.00	32	24·56	0·089	22·15	0·106
11.30	32	21·78	0·112	18·75	0·123

* In scale steps.

The concentration corresponding to scale step 0 for salt = log $\bar{3}$·10 = 0·0013 per cent.; for sweet = log $\bar{1}$·00 = 0·1000 per cent.; for sour = log 4·00 = 0·00010 per cent.; for bitter (Caffeine) = log $\bar{3}$·95 = 0·0089 per cent.

The three former of these studies are somewhat better controlled than the others in that their results are based on a greater number of threshold determinations under any one condition. Janowitz and Grossman's results are based on only one threshold determination at a given time of day. In Meyer's study the size of the step between successive concentrations in each series of stimuli was varied by a factor of two thus precluding the detection of variations of less magnitude than 100 per cent. Had finer gradations of stimuli been used a significant variation may well have been detected.

Goetzl *et al.* (1950) suggest that increases and decreases in sensitivity to sucrose are related to the sensation complexes of appetite and satiety respectively. Irvin and Goetzl (1952) make the same suggestion with respect to salt. The results obtained in the present study under fasting conditions do not support this suggestion. Invariably reports of increasing hunger were made towards the end of the 21-hr. fasting period, but in all cases the thresholds indicated a trend of a decrease in taste sensitivity after the first 16 hr. of fasting (i.e. after the 11.30 a.m. session). In other words, the period of increasing appetite was accompanied by a decrease in taste sensitivity. A similar effect is evident in the no-lunch results where the 4.45 p.m. thresholds are larger than the 2.15 p.m. thresholds. (Whilst the point was not rigidly checked, in general the spontaneous reports of the subjects indicated that they felt more hungry at 4.45 p.m. than at 2.15 p.m. during the "no lunch" days.)

This tendency for sensitivity to be less acute during the late afternoon than during the late morning is also present in the "normal" and "large" lunch results (i.e. under both of these conditions the 4.45 p.m. thresholds tend to be larger than the 11.30 a.m. thresholds).

This could be due to a fatigue factor. However, in later experiments on the influence of salt deprivation (to be reported elsewhere) it was found that the general

fatigue and feelings of lassitude associated with depletion of body salt did not have any generalized effects on the taste mechanism. In view of this finding it is felt that fatigue is probably not the factor responsible for the tendency of decreased sensitivity in the late afternoon.

The observed fluctuations do not appear to be directly related to the nutritional needs of the organism in any simple manner, for after some 22 hr. of fasting the thresholds for all taste qualities are higher than after about 6 hr. without food (i.e. the 4.45 p.m. "fasting" thresholds are higher than the 2.15 p.m. "no lunch" thresholds).

The intake of food appears to result in a fairly immediate decrease in sensitivity which is followed over the next 6 hr. with an increase in sensitivity. It also appears from the "normal lunch," "large lunch" and "no lunch" results that the degree of change in sensitivity, following food intake may be related to the caloric value of the food. The greater the caloric value of the meal the larger is the subsequent rise in the taste thresholds (i.e. the greater is the subsequent decrease in sensitivity). Note, however, that the relative degree of decrease and increase following breakfast is greater than that following a normal lunch or large lunch, even though the morning meal is of less caloric value than the midday meal (see Table I). This may be due to the different physiological reactions of the body to a meal which is preceded by some 12 hours of fasting (e.g. breakfast) as compared with those occurring after a meal preceded by about 4 hr. without food (e.g. lunch).

Further investigation is required to discover the nature of the variables underlying these systematic fluctuations in taste sensitivity. In the meantime it is evident that the gross factors of time of day and food intake need to be controlled in studies involving taste.

This investigation is part of research carried out by the author at the University of Cambridge for the Degree of Doctor of Philosophy. It was made possible by the grant of a Hackett Studentship from the University of Western Australia for which special thanks are due. The author is also indebted to Professor O. L. Zangwill, who supervised the research.

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SOME FACTORS AFFECTING TASTE SENSITIVITY IN MAN. II:

Depletion of Body Salt

BY

ROY YENSEN

*From the Psychological Laboratory, Cambridge**

Mal-function or surgical removal of the cortex of the adrenal glands leads to a severe loss of sodium chloride from the body. Adrenalectomized rats maintained on a normal diet usually die from excessive loss of salt in 8 to 15 days following the operation (Richter, 1942). Clark and Clausen (1943), Mark (1943), Richter (1936) and others have shown that adrenalectomized rats, when given free access to saline solutions, so increase their salt intake as to keep themselves alive and free from the symptoms of salt deficiency. It has also been shown that the preference threshold for solutions of sodium chloride is about 0·06 per cent. concentration in normal rats and about 0·004 per cent. concentration in untreated adrenalectomized rats (Bare, 1949; Richter, 1939). Similar findings are reported by Thomson and Porter (1953) for rats in a state of salt deficiency due to deprivation of salt from their normal diet.

That such preference behaviour is largely dependent on taste stimulation is suggested by Richter (1947) who states: "After sectioning of the taste nerves (bilateral section of lingual, chorda tympani and glossopharyngeal nerves) adrenalectomized rats no longer selected salt and died within a few days after the operation" (p. 131). Pfaffmann (1957) reports that "there is clear evidence of impairment in preference behavior following bilateral chorda tympani and IXth nerve removal. Taste stimulation thus appears to be an initial sensory instigator of such behavior" (p. 145).

Wilkins and Richter (1940) report the case of a three-year-old boy, who from the age of one year had eaten large amounts of salt literally by the handful. When his salt intake was restricted to the amounts present in the hospital diet, he rapidly developed symptoms of salt deficiency and died within a week. Autopsy revealed that a tumerous growth had destroyed the cortex of both adrenal glands. Richter (1942) states: "A salt craving is so often an early manifestation of Addison's disease that it is of considerable diagnostic significance" (p. 98).

As an explanation of the increased intake of salt by the salt-needy rat Richter (1939), Hartridge (1945) and Young (1949) have suggested that certain physiological needs lead to a change in taste sensitivity for the needed substance which in turn results in an increased intake of the required substance.

The present paper is concerned with the former assumption, namely, that partial depletion of body salt results in changes in taste sensitivity. This assumption does not follow from the results of preference studies with adrenalectomized rats, unless it is assumed that the preference threshold is either identical with or proportionally related to the absolute threshold. The latter has not been demonstrated.

In an endeavour to check this assumption Carr (1952) and Harriman and MacLeod (1953) used a conditioning technique to establish the rat's threshold of discrimination between water and salt solutions before and after adrenalectomy. In both studies

* Now at the School of Applied Psychology, University of New South Wales.

it is reported that adrenalectomy did not lead to a lowering of the threshold. Harriman and MacLeod concluded that "the thesis that salt homeostasis is maintained as a result of an automatic increase in *salt* sensitivity consequent upon salt deficiency is called into question. The broad fact of salt homeostasis is not challenged; but the nature of the behavioral support for salt homeostasis continues to be a question for research" (p. 471, italics by the present author). But this conclusion may not follow from the data reported. The discrimination learnt by the subjects in these studies is one of discrimination between salt solutions and water (Carr used tap water; Harriman and MacLeod used distilled water). It has been reported by Renqvist (1919), Richter and MacLean (1939), Cox and Nathans (1953), and noted by the present author, that human subjects can discriminate between water and salt solutions at concentrations definitely lower than those required to elicit a *salt* taste. At the discrimination threshold a wide variety of experiences are reported such as "sweetish"; "bitter"; "sour"; "smoother"; "a chemical taste." If such observations are relevant to the work of Carr and Harriman and MacLeod, then they have probably not gained data relating to absolute thresholds for salt, but to thresholds of discrimination between very low concentrations of salt solutions and water, which may be based on experiences other than a *salt* taste. The fact that Harriman and MacLeod report discrimination thresholds as low as 0·000025 per cent. concentration would tend to support the above suggestion.

A different approach to the problem was made by Pfaffmann and Bare (1950), who compared the strengths of the concentration of sodium chloride solutions just necessary to elicit an afferent impulse in the chorda tympani nerve of sham adrenalectomized and bilateral adrenalectomized rats. For both groups the critical concentration was at approximately the same value. They concluded "that salt deficiency does not alter the sensitivity of the taste receptors" (p. 324). However, this conclusion is based on neural records sampling activity in the chorda tympani nerve only and so not on the total sensory input from the taste field. In the second place, even if such records are a truly representative sample of the total gustatory input, they do not exclude the possibility of changes in taste sensitivity of a central rather than peripheral nature. In fact, in a more recent article Pfaffmann (1957) concludes that the changes in preference-aversion behaviour "under conditions that modify the physiologic state of the organism . . . reflect not a change in the peripheral afferent neural message but changes in its significance for central neural processes" (p. 147).

To-date, then, the hypothesis that partial depletion of salt in the body is accompanied by changes in taste sensitivity has not been adequately checked. The present paper reports the results of an experiment designed for such a check.

The use of animal subjects can only yield preference thresholds or thresholds of discrimination between a given solution and water, which (as noted above) do not necessarily indicate the absolute threshold for the given taste quality. For this reason human subjects were used in the present study.

Taste sensitivity is assessed in terms of the absolute threshold for taste of the accepted four basic taste qualities of sweet, salt, sour and bitter (Geldard, 1953; Moncrieff, 1951; Pfaffmann, 1951). Details of the method used to determine the taste threshold were presented in a previous report (Yensen, 1959) and will not be repeated here.

Procedure

Two experiments were carried out with a break of 6 weeks between them. In the first experiment taste thresholds for salt and sour were determined daily over 21 days. The first 6 days were used as a control period, the next 8 days as an experimental or treat-

ment period and the final 7 days as a recovery period. In the second experiment thresholds for salt, sweet, and bitter (quinine sulphate) were determined over 18 days, made up of periods of 6 days control, 7 days treatment and 5 days recovery. Two subjects, H and R, were used in each experiment. Four threshold determinations were made during each session.

During the control periods the subject was in normal salt balance. The treatment periods were designed to bring about a gradual increase in losses of body salt which were corrected during the recovery periods. Thus each subject acted as his own control. Test conditions were standardized over the full period of these experiments. The general routine, the caloric value and composition of the daily food intake and the test procedures were the same as used in the experiments on diurnal variations (Yensen, 1959) except for certain changes during the treatment periods. Water was allowed as desired during both experiments.

A procedure similar to that reported by McCance (1936) was used to induce losses of body salt. It consisted of considerable reduction in the daily salt (sodium chloride) intake, combined with forced loss of salt in five $1\frac{3}{4}$-hr. and four 2-hr. sweating sessions during the first and second treatment periods respectively. These sessions were run after the day's testing was finished (i.e. late afternoon) in a hot room at 95° F. wet bulb and 105° F. dry bulb. Subject R sweated more readily and freely than subject H, who found it necessary to do some exercises to hasten the onset of marked sweating. This usually started after about 10 min. and then tended to keep up throughout the session. During the second treatment period both subjects tended to sweat a little more readily and freely. Apart from tiredness and an occasional headache these sessions had no upsetting effects.

During the control periods the salt intake of both subjects was approximately 10 gm./day. By manipulation of the diet this "normal" intake was reduced to an average of 78 mgm. sodium and 102 mgm. chloride/day for subject H, and to 80 mgm. sodium and 103 mgm. chloride/day for subject R during the treatment period of the first experiment. Corresponding figures for the second experiment were 72 mgm. sodium and 85 mgm. chloride/day for subject H, and 75 mgm. sodium and 88 mgm. chloride/day for subject R. The subjects were weighed under standard conditions (i.e. about 10.15 a.m. with empty bladder and in the same dress without shoes or heavy outer clothing) on various days throughout both experiments. Losses in body weight of 1·800 kgm. and 1·550 kgm. for subject H, and 1·750 kgm. and 2·100 kgm. for subject R were recorded over the first and second treatment periods respectively.

TABLE I

SERUM-SODIUM AND PLASMA-CHLORIDE CONCENTRATIONS DURING THE SECOND EXPERIMENT ON SALT DEPRIVATION. SUBJECTS H AND R*

Period	*Day of period*	*Serum-sodium in mEq./l.*		*Plasma-chloride in mEq./l.*	
		H	*R*	*H*	*R*
Control	last	143	140	109	108
Treatment	3rd	135	138	106	104
,,	5th	134	136	103	102
,,	last	133	138	105	101
Recovery	1st	141	140	111	109
,,	3rd	144	142	110	109

* Thanks are due to Prof. R. A. McCance, Department of Experimental Medicine, University of Cambridge, for arranging the taking and analysis of these samples.

During the second experiment venous blood samples were taken and analysed through arrangement with Professor R. A. McCance of the Department of Experimental Medicine, University of Cambridge. The serum-sodium and plasma-chloride concentrations of these samples are shown in Table I. Samples were preserved until all were collected and they were then analysed at the same time. Silver nitrate tests at the end of each treatment period showed the urine of both subjects to be practically clear of chloride.

During the last 3 days of the treatment periods, both subjects reported feelings of increasing tiredness and heaviness of the limbs. Even slight physical exertion, such as walking up a flight of stairs, was extremely tiring and accompanied by shortness of breath. Concentration and motor co-ordination also seemed to be slightly impaired. Despite the drinking of large amounts of water, both subjects experienced considerable dryness of the mouth and thirst, especially over the last 2 days. Polyuria was present during this time. No marked disturbances of sleep occurred at any stage in either subject. Subject R experienced occasional spasms of cramp in the leg muscles during the last 2 days of the first treatment period.

Neither subject found great difficulty in eating the special diet, except that both found the salt-free bread rather unpleasant. It tasted sweetish. Other foods which are normally eaten with salt tasted unusual, but not noticeably different from the way they taste without salt under normal conditions. For example, potatoes tasted slightly bitter with a trace of sweet; beans slightly sweet and sour and stewed beef rather flat, but with a peculiar "meaty" taste. Foods which are normally eaten without added salt (e.g. apples, plums, honey, sugar, roasted peanuts) did not taste different. Apart from the feeling at meal times that some of the food would probably "taste nicer with some salt" no cravings for salt were felt by either subject.

In the first experiment recovery was relatively slow. Salt intake was restricted to about 7½ gm. per day for the first 4 days of the recovery period. After this, normal salt intake (about 10 gm. per day) was allowed. Despite the drinking of a considerable amount of water both subjects had recurring thirst during the first day or so, over which the polyuria declined. The feelings of extreme fatigue lessened gradually and by the fourth day both subjects felt completely recovered.

Recovery in the second experiment was quite rapid. The salt intake was about 22 gm. per day over the first 2 days, 18 gm. on the third day, after which normal intake was resumed. Each subject took 10 gm. of salt with the first meal of this period. It led to almost immediate relief of the feeling of heaviness in the arms and legs and noticeable lessening of the fatigue which both subjects had experienced over the preceding days. Acute thirst was present despite considerable drinking and the passing of very small volumes of urine over the first day. This marked retention of water following the high salt intake is reflected in the increases in body weight in both subjects. In the 20 hr. following this first salt meal subject H gained 2·05 kgm. and subject R 2·3 kgm.

It is evident from the following points that both salt depletion and a mild state of salt deficiency were induced during the later stages of the treatment periods. (Salt depletion refers to a reduction in the total salt content of the body; salt deficiency refers to a drop in the sodium concentration in the extracellular fluids (Peters, 1952).)

(1) The low intake of salt: an average of less than 0·2 gm. NaCl per day over these periods.
(2) The renal output of salt over the first few days would undoubtedly be much greater than the input.
(3) The additional extra-renal losses of salt through sweating.
(4) The symptoms reported by the subjects.
(5) The losses in body weight, despite the constancy of the caloric value of food intake and the drinking of large amounts of water.
(6) The extremely low chloride content of the urine at the end of the treatment periods.
(7) The fairly rapid gains in body weight following the increases in salt intake during the recovery periods.
(8) The fall and rise in the serum-sodium and plasma-chloride concentrations during the treatment and recovery periods of the second experiment.

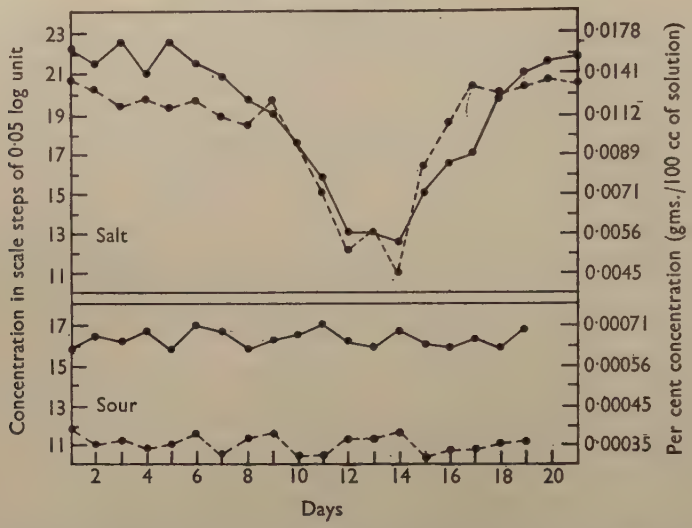
Results

The mean thresholds per session for salt and sour during the first experiment are shown in Figure 1, and those for salt, sweet and bitter during the second experiment are shown in Figure 2.

Let us first consider the thresholds for sour, bitter, and sweet. One-way analyses of variance of these values show no significant differences ($P > 0{\cdot}05$) between the control, treatment and recovery periods. It appears, then, that sensitivity to sour, bitter and sweet was not affected during these experiments.

It is obvious from Figures 1 and 2 that this is not so for salt. The results of *t*-tests between the control period thresholds and those of the last 4 days of the treatment periods show a highly significant difference (Table II). In other words, for both subjects taste sensitivity to salt significantly increased (i.e. the threshold decreased) during the treatment periods of these experiments.

FIGURE 1



Mean daily thresholds for "salt" and "sour" during the "control," "treatment" and "recovery" periods of the first experiment. Subject H: ———; Subject R: - - - - .
The concentration corresponding to scale step 0 for salt = $\log \bar{3}{\cdot}10 = 0{\cdot}0013$ per cent.; for sour = $\log \bar{4}{\cdot}00 = 0{\cdot}00010$ per cent.

TABLE II

t-RATIOS BETWEEN THE "SALT" THRESHOLDS OF THE "CONTROL" PERIOD AGAINST THE LAST 4 DAYS OF THE "TREATMENT" PERIOD OF EACH EXPERIMENT. SUBJECTS H AND R

		H		*R*	
Source	*Experiment*	*Difference in scale steps*	*t-ratio**	*Difference in scale steps*	*t-ratio**
Control v. 4 days Treatment ..	1	−8·06	22·38	−6·86	17·59
	2	−9·32	38·83	−7·88	37·52

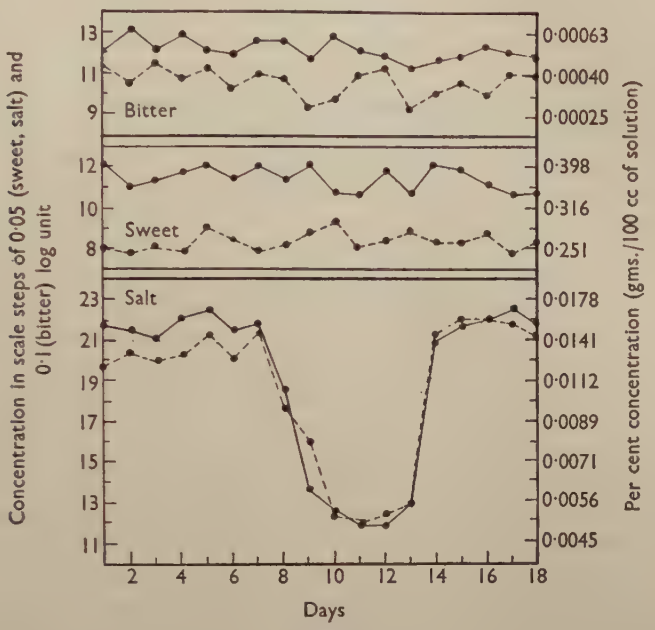
* All significant beyond 1 per cent. level. d.f. = 38.

The reliability of the threshold determinations in these experiments is indicated by the very low standard errors of the means shown in Table III.

From time to time at the end of a run during the salt sessions in the treatment periods, the subjects were presented with salt solutions which had been around threshold concentration under normal conditions. Their spontaneous responses to these stimuli were recorded. This qualitative check revealed that salt solutions

which were mildly salty under normal conditions were stronger and quite definitely salty under conditions of increased sensitivity to salt.

FIGURE 2



"Mean daily thresholds for "bitter," "sweet" and "salt" during the "control," "treatment" and "recovery" periods of the second experiment. Subject H: ———; Subject R: - - - - - .

The concentration corresponding to scale step 0 for bitter (quinine) = log $\bar{5}\cdot50$ = 0·000032 per cent.; for sweet = log $\bar{1}\cdot00$ = 0·10000 per cent.; for salt = log $\bar{3}\cdot10$ = 0·0013 per cent.

TABLE III

MEANS, AND STANDARD ERRORS OF THRESHOLD MEASUREMENTS. SUBJECTS H AND R

Subject	*Source*	*No. of det.*	*Mean in scale steps*	*S.E. (of mean)*
H	Salt, Control, Experiment 1	24	21·87	0·178
	Sour, Experiment 1	76	16·29	0·070
	Sweet, Experiment 2	72	11·50	0·092
	Bitter, Experiment 2	72	12·29	0·078
	Salt, Control, Experiment 2	24	23·75	0·161
R	Salt, Control, Experiment 1	24	19·79	0·153
	Sour, Experiment 1	76	10·01	0·075
	Sweet, Experiment 2	72	8·28	0·074
	Bitter, Experiment 2	72	10·58	0·104
	Salt, Control, Experiment 2	24	20·25	0·137

The concentration corresponding to scale step 0 for salt = log $\bar{3}\cdot10$ = 0·0013 per cent.; for sweet = log $\bar{1}\cdot00$ = 0·1000 per cent.; for sour = log $\bar{4}\cdot00$ = 0·00010 per cent.; for bitter (quinine) = log $\bar{5}\cdot50$ = 0·000032 per cent.

Discussion

Since no effect on the sour, bitter or sweet thresholds was apparent, it appears that the treatment conditions had no generalized influence on the taste mechanism. This suggests that the definite fatigue and other symptoms experienced during the later stages of the treatment periods had no influence on taste sensitivity.

Comparison of the rates of increase in sensitivity to salt during the first and second treatment periods shows a tendency for the rate to be greater during the latter period. Salt intake per day was slightly less and extra-renal output probably greater during the second treatment period in which the sweating sessions were more successful and longer (i.e. the salt losses were probably greater during the early stages of the second treatment period). It is suggested that the more rapid increase in sensitivity was due to the greater losses of salt during the earlier stages of the second treatment period.

There is also a marked difference in the rates of return to normal threshold levels between the first and second recovery periods. Near normal levels were reached and maintained some 24 hr. after the increases in salt intake during the second experiment, whereas in the first experiment return to normal levels took much longer. Over the first day of the second period the salt intake was considerably larger (22 gm. as against $7\frac{1}{2}$ gm.) and the relief from symptoms more complete than over the first day of the first recovery period. The increases in body weight and the return to normal levels in the serum-sodium and plasma-chloride concentrations over the first day of the second recovery period suggest that a considerable proportion of the 22 gm. of salt was retained. It is suggested that the more rapid return to normal threshold levels (i.e. decrease in sensitivity) was due to the quicker correction of the salt losses during the second recovery period.

These differences between the rates of increases and decreases in sensitivity during the two experiments suggest that, within limits, sensitivity to salt may be related to the degree of salt losses. This suggestion is also supported by the following observation. It is fairly reasonable to assume that there were progressive increases in the amount of total salt losses over the first half of the treatment periods. During this time both subjects showed progressive increases in sensitivity to salt.

Some of the factors which varied during the treatment periods will now be considered.

In a healthy individual, a loss of salt is accompanied by a loss of body water, even though his intake of water is normal. In these experiments both subjects showed losses in body weight during the treatment periods. Since the caloric value of the food intake was constant throughout the experiments, the rather rapid fluctuations in body weight can be taken as indications of changes in the volume of body water. Thus, despite the relatively large intake of water, both subjects experienced losses in body water as well as losses in body salt.

The losses in body water are due to a reduction in the volume of the extracellular fluid. This tends to counteract the fall in the level of concentration of sodium in these fluids, until a point is reached where further reduction in volume is presumably more upsetting to the organism than further falls in sodium concentration. So a reduction in the volume and in the osmotic pressure of the extracellular fluid occur during a state of salt deficiency. It could be that either one of these variables, or an interaction between them, was responsible for the changes in sensitivity to salt found in both subjects in the present experiments.

An indication of the importance of each of these variables may be gained from a study of sensitivity to salt during a period of water deprivation. Under this condition there is a tendency for the volume of the extracellular fluid to fall (the main

loss of water is from intracellular fluid) accompanied by a slight rise in the sodium concentration of the extracellular fluid. If a reduction in the volume of the extracellular fluid is the variable underlying changes in sensitivity to salt, then a period of water deprivation should result in increases in sensitivity to salt.

There was another variable present during the treatment periods, namely a very drastic reduction in the amount of salt stimulation of the taste receptors. The very small quantities of salt which were present in the food were not sufficient to elicit a distinct salt taste during eating. The amount of salt stimulation of the taste receptors during threshold determinations was extremely small (i.e. at the most ten salt solutions around threshold levels were tasted daily). It is possible that this lack of stimulation may have been responsible for the observed changes in sensitivity to salt. If this is so, then sensitivity to salt should not vary during a period of water deprivation, provided normal use of salt is allowed.

Before entering on further discussion of the results presented in this paper, it seems desirable to ascertain which of the three variables outlined above most probably underlies the observed changes in sensitivity to salt. Details of an experiment designed to check the influence of depletion of total body water on taste sensitivity will be presented in a further communication in which the present results will be more fully discussed.

One point remains to be stressed, namely that from the results gained in this study and in a previous report (Yensen, 1959), it appears that the taste mechanism is of such a nature that taste sensitivity is influenced by certain changes in the physiological state or "milieu interieur" of the organism. Especial care needs to be exercised to ensure control of these variables in all studies involving taste.

This investigation is part of research carried out by the author at the University of Cambridge for the Degree of Doctor of Philosophy. It was made possible by the grant of a Hackett Studentship from the University of Western Australia for which special thanks are due. The author is also indebted to Professor O. L. Zangwill who supervised the research.

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SOME FACTORS AFFECTING TASTE SENSITIVITY IN MAN. III:

Water Deprivation

BY

ROY YENSEN

*From the Psychological Laboratory, Cambridge**

In a previous article (Yensen, 1959*b*) it was shown that forced loss of salt from the body due to a very low salt intake and sweating, is accompanied by an increase in taste sensitivity to salt, but that no changes occur in sensitivity to sweet, sour or bitter. It was suggested that the observed change in sensitivity to salt may be due to a reduction in the volume of the extracellular fluid; to a lowering of the sodium concentration of these fluids; or, to a lack of salt stimulation of the taste receptors. The relative importance of these three variables might be ascertained from a study of taste sensitivity during a period of water deprivation.

A study relevant to this problem is that of Young and Falk (1956), who report on the preference behaviour of thirsty (water deprived for 17½ to 19 hr.) and non-thirsty rats towards salt solutions. They found that non-thirsty rats showed definite preferences for salt solutions up to 0·75 to 1·5 per cent. concentration and some acceptance of hypertonic solutions. Thirsty rats completely rejected hypertonic solutions and tended to prefer water rather than any of the weaker salt solutions. Salt deficient rats exhibit marked increases in their preferences for both hypertonic and hypotonic salt solutions. Thus it may be said that the preferences shown by water-deprived rats towards salt solutions are the direct opposite of those shown by salt deficient rats. Since it has been shown that depletion of body salt is accompanied by an increase in taste sensitivity to salt, it might be expected on the basis of Young and Falk's results that depletion of body water will be accompanied by a decrease in sensitivity to salt.

The present paper reports the results of an experiment designed to check this prediction.

Details of the method used to determine taste thresholds were presented in a previous report (Yensen, 1959*a*). A slight modification to that method was introduced in this study. During preliminary practice sessions subjects were instructed to develop a method of rinsing and drying the mouth which reduced to a minimum the amount of fluid swallowed during testing. This was achieved by the subject spitting out as much as possible of the fluid remaining in the mouth after rinsing. Nevertheless, some very small amounts of fluid were undoubtedly swallowed during testing.

Procedure

Since it appears that food intake influences taste sensitivity (Yensen, 1959*a*), it is essential that this factor be controlled, as much as possible, during the period of water deprivation. This means that fluid intake cannot be completely eliminated. However, it can be drastically reduced by choosing a diet with a very low water content and refraining from any fluid intake apart from the amounts present in the food and released during oxidation.

* Now at the School of Applied Psychology, University of New South Wales.

During a period of 2 to 3 days under such conditions a gradual loss of body water is inevitable. Compensatory reduction in the "normal" volume of urine (i.e. about 2,000 c.c. per day) occurs fairly rapidly, but this will still be about 300 to 400 c.c. per day by the third day. Little or no change occurs in the insensible losses from the skin and lungs which are "usually of the order of 1,000 c.c. per day" (Black *et al.*, 1944, p. 410). Losses through sensible perspiration may also occur depending on the subject's activities and climatic conditions. The volume of both the intracellular fluid and extracellular fluid is reduced and the osmotic pressure of the body fluids rises (Fulton, 1955).

Four subjects were used: H and R, who participated in the previous experiments (Yensen, 1959*a* and *b*); subject J, a 22-year-old male research student; and subject C, a 20-year-old male undergraduate. The experiment was run over 9 days each with subjects H and R and 8 days each with subjects J and C. The first 3 days were used as a control period under conditions of normal food and water intake for all subjects. This was followed by a treatment period under conditions of a diet of low water content and restricted water intake. Finally, there was a recovery period in which normal food and free intake of water were allowed.

Two sessions for determining thresholds for salt and one session for sour were conducted daily with each subject. Four determinations were made in each session. Subjects H and R were tested for salt thesholds at 9 a.m. and 4.45 p.m. and for sour at 2.15 p.m. To fit in better with their normal duties, subjects J and C were tested at 9 a.m. and 11.30 a.m. for salt and at 2.30 p.m. for sour thresholds. The two sessions per day on salt were included to see whether the variations in sensitivity found in earlier experiments on diurnal variation would also occur under conditions of water deprivation.

Throughout the experiment the caloric value and the times of meals were kept constant as shown in Table I. The caloric values approximate the food intakes of the subjects under normal conditions. The composition of the daily food intake varied slightly during the treatment period as compared with the control and recovery periods. In order to keep the proportion of protein comparable and the food itself reasonably acceptable, the proportion of carbohydrate was slightly reduced and that of fat slightly increased. In general, the dry diet consisted of "Ryvita," digestive biscuits, cream crackers, butter, cheese, milk chocolate, bacon and dates. Although the intake of salt was not restricted, it was probably slightly less than normal during the treatment period.

TABLE I

Caloric Values* and Times of Meals

Meal	*Times*				*Calories*			
	H	*R*	*C*	*J*	*H*	*R*	*C*	*J*
Breakfast (a.m.)	7·45	7·45	8·00	7·45	510	760	650	840
Morning tea (a.m.)	10.15	10.15		10.30	85	125		50
Lunch (p.m.)	12.30	12.30	12.40	12.40	660	810	860	900
Afternoon tea (p.m.)	3.15	3.15	4.00	4.30	85	125	65	150
Dinner (p.m.)	7.00	7.00	5.45	7.00	760	830	875	900
	Total calories per day				2,100	2,650	2,450	2,840

* Calculated from McCance, R. A., and Widdowson, E. M. (1946). *The Chemical Composition of Foods.* 2nd Ed. London.

The procedures during the treatment periods differed for subjects H and R from those used for subjects J and C. The hot room used for sweating sessions by subjects H and R was demolished before either of the latter subjects was available.

For subjects H and R the period of water deprivation started at 8.30 a.m. on the fourth day and lasted until 11.30 a.m. on the sixth day of testing. A 2-hr. sweating session was carried out in a hot room at 95° F. wet bulb and 105° F. dry bulb from 10.30 a.m. to 12.30 p.m. on the first day of the treatment period. Both subjects sweated freely during most of this session. Apart from the very small quantities which may have been swallowed during testing, no fluid was taken during this period of 51 hr. Over this period subject H lost 2·500 kgm. and subject R. lost 3·150 kgm. in body weight. The total water intakes

from food and oxidation during the treatment period were 659 c.c. for subject H and 834 c.c. for subject R. Samples of venous blood were taken on the days shown in Table II which also shows the serum-sodium and plasma-chloride concentrations and the haematocrit percentages on these days.

During the treatment period both subjects noted a considerable reduction in the volume of urine passed during the first day. On the second day and morning of the third day only very small amounts were voided. Both experienced progressive increases in dryness of the mouth which became extreme during the later stages. The chewing of gum relieved this during the earlier part, but was later ineffective. Increasing tiredness and irritability were also reported, although sleep was not noticeably disturbed on either of the two nights during this period. On the first day the desire to drink was not very strong, but this gradually increased to a very strong desire in both subjects towards the end of the period. The first few meals of dry food presented no great difficulties, but subsequently they were rather unpalatable and at times slightly nauseating. Both subjects found it extremely difficult to eat the full amount prescribed for the last two meals and, in fact, at breakfast on the final day both left a small amount (about 1 oz. of Ryvita each). Swallowing the dry food was difficult and most unpleasant. It was necessary to rinse the mouth after meals to rid it of food particles.

At the beginning of the recovery period (i.e. 11.30 a.m. on the sixth test day) subject H drank two large glasses of water straight off and a third soon after which greatly relieved her thirst. Between this time and 9 a.m. on the following day she drank a total of about 3½ litres of fluid. Subject R drank three large glasses of water immediately and felt considerably relieved. By 9 a.m. on the following morning he had taken nearly 5 litres of fluid. The urine volumes of both subjects were "about normal" on the first morning of the recovery period.

The treatment period for subject C started at 6 p.m. on the third test day and lasted until 12 p.m. on the sixth test day. No fluid was deliberately taken during this period of 66 hr., over which he lost 2·900 kgm. in body weight. The total intake of water from food and oxidation was 837 c.c. The results of analysis of venous blood samples taken during the experiment are shown in Table II. In general, the experiences of subject C during the treatment period were similar to those of subjects H and R. However, he did not find the dry diet quite so objectionable. At the beginning of the recovery period he drank 3 glasses of water and by 9 a.m. the next morning had taken nearly 4 litres of fluid and felt "quite as usual again."

TABLE II

CONCENTRATIONS OF SODIUM IN SERUM, CHLORIDE IN PLASMA AND HAEMATOCRIT PERCENTAGES*

Day of experiment	*Serum-sodium (m.equiv./l.)*		*Plasma-chloride (m.equiv./l.)*		*Haematocrit (per cent.)*	
	R	*H*	*R*	*H*	*R*	*H*
3	147	147	109	112		
6	160	155	118	115	48·7	44·9
7	148	146	107	111	48·4	44·8
9	148	144	108	110	46·5	40·9
	J	*C*	*J*	*C*	*J*	*C*
3	144	137	109	104	50·1	45·1
6	147	141	111	113	51·9	46·7
8	144	141	110	108	49·2	46·1

* Thanks are due to Prof. R. A. McCance, Department of Experimental Medicine, University of Cambridge, for arranging the taking and analysis of these samples.

The first attempt to run the experiment with subject J had to be abandoned. Towards the end of the first day of water deprivation he developed a headache and a body temperature of 101° F. and felt rather unwell. Since it was unlikely that these symptoms were

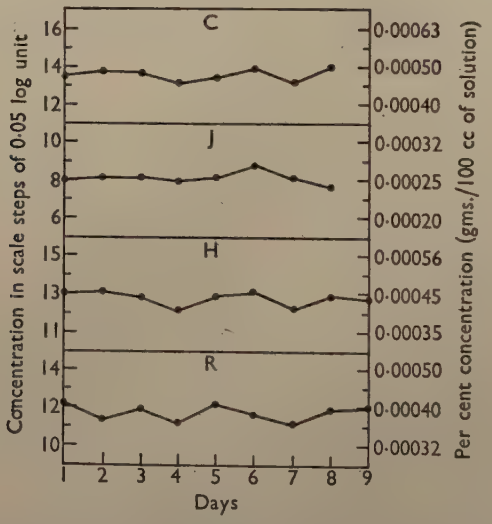
due to some 16 hours of water deprivation, medical advice was sought and the subject was rehydrated. He felt off colour for a few days, but agreed to a repeat of the experiment about a week later.

After practice testing and a 3-day control period subject J began his second treatment period at 9 p.m. on the third test day and refrained from deliberate fluid intake until 10.30 a.m. on the sixth test day. (Subjects C and J were not continually supervised during the treatment periods, but apart from other considerations, the recorded losses in body weight and the observed changes in blood samples strongly support the author's belief that they faithfully carried out the experimental instructions.) During this period of 61½ hr. J lost 2·250 kgm. in body weight. His total intake of water from food and oxidation was 1,000 c.c. The results of analysis of venous blood samples taken during the experiment are shown in Table II. His experiences of thirst, dryness of the mouth and decreases in urine volume over the treatment period were similar to those of the previous subjects. He found the dry diet unpleasant and difficult to swallow, but "managed to eat" all of it, except half a digestive biscuit at breakfast on the fifth test day. J's sleep was disturbed ("woke up fairly frequently feeling very thirsty") and he reported feeling that his "efficiency was slightly impaired" during the treatment period. However, in general he felt "no outstanding symptoms." At the beginning of the recovery period J drank four cups of water straight off and felt "much better." By 9 a.m. on the next morning he had taken about 4 litres of fluid and felt "quite all right this morning."

That losses in body water were experienced by all subjects during the treatment periods is evident from the following points:

(1) The very low intakes of water during this period.
(2) The losses and gains in body weights during the experiment.
(3) The changes in blood composition (Table II).
(4) The symptoms experienced by the subjects.

FIGURE 1



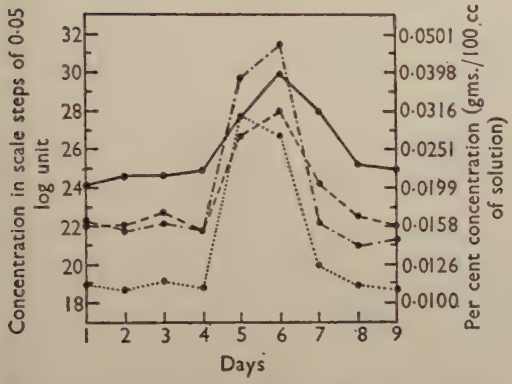
Mean daily thresholds for "sour" during the "control," "treatment" and "recovery" periods. The concentration corresponding to scale step 0 for sour = log $\bar{4}$·00 = 0·0001 per cent.

Results

The mean daily thresholds for sour of each subject are shown in Figure 1. Analyses of variance of the results show no significant variation between the control, treatment and recovery periods ($P > 0·05$).

The mean thresholds for salt at each session are presented in Figures 2 and 3. The threshold of all subjects showed marked increases during the treatment period. The results of *t*-tests between the 9 a.m. thresholds of the control periods and the last two days of the treatment periods show these differences as highly significant ($P < 0.01$) for all subjects. It appears, then, that the periods of water deprivation resulted in significant decreases in sensitivity to salt for all subjects.

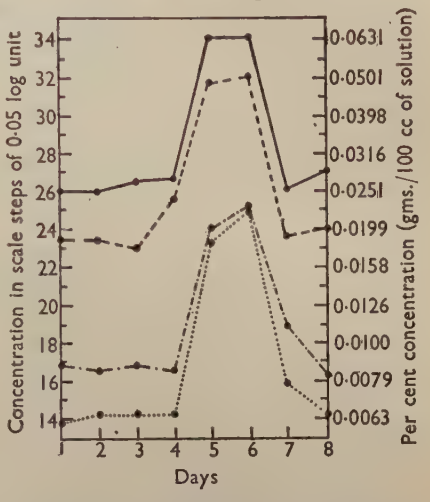
FIGURE 2



Mean daily thresholds for "salt" during the "control," "treatment" and "recovery" periods. Subject H: ——— 9 a.m., - - - - 4.45 p.m.; subject R: —.—.— 9 a.m.; ..... 4.45 p.m.

The concentration corresponding to scale step 0 for salt = log $\bar{3}$·10 = 0·0013 per cent

FIGURE 3



Mean daily thresholds for "salt" during the "control," "treatment" and "recovery" periods. Subject C: ——— 9 a.m.; - - - - 11.30 a.m.; subject J: —.—.— 9 a.m.; ..... 11.30 a.m.

The concentration corresponding to scale step 0 for salt = log $\bar{3}$·10 = 0·0013 per cent.

The control period results show a significant difference ($P < 0.01$) between the 9 a.m. and the 11.30 a.m. thresholds for subjects J and C and between the 9 a.m. and 4.45 p.m. thresholds for subjects H and R. This finding supports earlier results (Yensen, 1959*a*) showing a significant variation in taste sensitivity during a day of normal food intake.

This variation with time of day has persisted during the treatment and recovery periods over which in every case the 9 a.m. thresholds are larger than those taken later in the same day. As Figures 2 and 3 show, the size of this variation has not remained constant during these periods. This is probably due to the fact that the tendency for the threshold to decrease from the 9 a.m. to the later session was partly counteracted by the tendency for it to increase due to the increasing loss of body water. It appears, then, that the variations due to food intake were still present, even when the level of the threshold had increased markedly during the treatment periods. This suggests that the factors behind the influence of food intake which occurs over all four taste qualities, are independent of the factors causing the above changes in sensitivity to salt.

An indication of the reliability of the threshold determinations in these experiments may be found in Table III.

TABLE III

MEANS, AND STANDARD ERRORS OF THRESHOLD MEASUREMENTS

	9·00 "*Control*" *salt*			*Sour*		
Subject	*No. det.*	*Mean**	*S.E. (of mean)*	*No. det.*	*Mean**	*S.E. (of mean)*
H	12	24·58	0·148	36	12·89	0·099
R	12	22·08	0·148	36	11·81	0·096
C	12	26·17	0·141	32	13·62	0·097
J	12	16·66	0·166	32	8·22	0·120

* In scale steps.

The concentration corresponding to scale step 0 for salt = log $\bar{3}$·10 = 0·0013 per cent., and for sour = log $\bar{4}$·00 = 0·00010 per cent.

DISCUSSION

Let us now compare the influence on taste sensitivity of the depletion of body salt with that of the depletion of body water.

The former results in an increase and the latter in a decrease in sensitivity to salt, whilst in both cases sensitivity to the other taste qualities remains unchanged.

In a previous article (Yensen, 1959*b*) it was suggested that the observed change in sensitivity to salt which accompanied depletion of body salt may have been due to a lack of stimulation of the salt receptors during the treatment periods. However, this factor remained fairly constant throughout the experiment on water deprivation in which decreases in sensitivity occurred. Thus, although it is not conclusive, it appears likely that the observed changes were not due to this factor.

During both experiments losses in body water occurred, but there are important differences in some of the accompanying changes in the distribution and tonicity of body water.

As depletion of the total salt in the body occurs, there is a reduction in renal output of salt and a tendency develops for the osmotic pressure of the intravascular system to fall. The latter leads to a reduction in the secretion of the antidiuretic hormone (ADH) from the neurohypophysis which results in a decrease in tubular reabsorption of water, thus leading to a slight reduction in the volume of the extracellular fluid. This tends to keep up the tonicity of the extracellular fluid. With further salt losses the above changes recur until the volume of the extracellular fluid is no longer sacrificed in the interest of maintaining osmotic pressure which then permanently falls if the salt losses continue, i.e. salt deficiency occurs. As hypotonicity of the extracellular fluid develops there is some movement of water from this compartment into intracellular spaces due to the differences in tonicity.

In the early stages of a period of water deprivation the normal renal and extra-renal outputs of water occur. These result in slight decreases in the volume of extracellular fluid and slight increases in its tonicity. The latter leads to an increase in the secretion of ADH which results in a decrease in the volume of urine passed. There is also a tendency for water to move from intracellular to extracellular compartments, thus tending to correct the developing hypertonicity of the extracellular fluid. However, the extra-renal losses, mainly from the skin and lungs, continue at about their normal level and small volumes of concentrated urine are also passed. Thus, as the period of water deprivation is prolonged, further reduction in the volume of the extracellular fluid and increases in its osmotic pressure occur, which tend to be

counteracted by the movement of water from the cells into the extracellular fluid. The final picture after 2 to 3 days is a definite reduction in the volume of the intracellular fluid, a slight reduction in the volume of the extracellular fluid and a rise in the osmotic pressure of both. (This brief summary is based on McCance, 1936; Black *et al.*, 1944; Gamble, 1950; Peters, 1952; Fulton, 1955.)

Thus in both experiments there were reductions in the volume of the extracellular fluid. Since the changes in sensitivity to salt were in opposite directions during these experiments, it would appear likely that the changes in volume per se of the extracellular fluid were not the main cause of the changes in sensitivity to salt. On the other hand, the tendencies for the sodium concentrations in the extracellular fluid to fall during salt deprivation and to rise during water deprivation parallel the changes which occurred in salt thresholds. This suggests that there may be a relationship between the sodium concentration of the extracellular fluid and sensitivity to salt.

It is interesting to consider the probable function of these changes in sensitivity to salt as part of the organism's reactions to variations in its physiological state. Under the conditions in which these sensitivity changes occurred the initial stimulus to some of the physiological reactions of the organism was the tendency for the osmotic pressure of the extracellular fluid either to fall (forced loss of salt) or to rise (loss of water). One of the main effector organs for the physiological reactions which result from such changes in the osmotic pressure of the extracellular fluid is the kidneys. However, as Robinson (1954) states: "The kidneys can only regulate the osmotic pressure of the extracellular fluids if the diet provides an adequate intake of water and sodium salts" (p. 121). Since the kidney can only regulate effectively if the dietary intake is adequate, there would presumably be little biological value from an evolutionary viewpoint in the development of such a complex organ unless there was also some mechanism which could influence the intake of water and sodium salts. Following losses of salt the intake of salt obviously needs to be increased. After losses of body water the intake of water needs to be increased, and, under normal conditions, the thirst mechanism brings this about. However, if water is not available, further salt intake would appear undesirable. For to check further rises in osmotic pressures of the extracellular fluid the additional salt has to be excreted and this usually requires the excretion of additional amounts of water, which the organism is already striving to conserve.

It is suggested, then, that the observed changes in sensitivity to salt are probably part of a mechanism which, in the absence of overriding psychological factors, would regulate the intake of salt.

That such a mechanism does exist is suggested by the results of work on rats. Untreated adrenalectomised and sodium deprived rats show marked increases in their intake of salt (Richter, 1936; Bare, 1949; Thomson and Porter, 1953; Epstein and Stellar, 1955). They drink more from all concentrations of salt solutions, ranging from about 0·003 to 6 per cent., than do normal rats. (As a result of this the preference threshold of the salt-needy rat is lower than that of the normal rat, e.g. 0·003 per cent. adrenalectomized, 0·055 per cent. normal: Richter, 1939*a*.)

Carpenter (1956) states that "water deprived rats drink less NaCl solutions" (p. 143). Young and Falk (1956) have shown that "thirsty rats completely reject salt solutions of 3·0 and 6·0 per cent., but non-thirsty animals occasionally accept them" (p. 575). Stellar *et al.* (1954) state: "Dehydrating animals by water deprivation will have the same effect on the preference-aversion curve as will hypertonic solutions introduced into the stomach—i.e. elevating the drinking of water and hypotonic solutions, and depressing the drinking of hypertonic solutions" (p. 224). Adolf (1943) found that rats deprived of water for 1 to 2 days drank very little when

given 24 hr. access to hypertonic salt solutions, but when given hypotonic solutions they drank about twice as much as when water was offered. He states: "By ingesting double the usual volume, they thus paid off the debt of body water and at the same time were able to eliminate the salt taken in this drink" (p. 29). Thus, after losses of body salt, the rat often increases its intake of salt and decreases it following losses of body water. Weiner and Stellar (1951) report that the preference-aversion behaviour of normal rats does not depend on previous experience with salt and that it shows up in the rate of drinking within the first 5 min. of contact with the most preferred solution. Epstein and Stellar (1955) have shown that the increased intake of the adrenalectomized rat is not dependent on post-operative experience with salt. Both Richter (1939*b*) and Pfaffmann (1952) have shown that the preference behaviour to salt solutions is considerably impaired by partial denervation of the taste receptors.

It appears, then, that these changes in intake of salt are dependent on some innate receptor-effector mechanism in which taste stimulation plays a very important role. From the experimental results of the present study and the results of work on animals, it would appear that the mechanism is such that increases in sensitivity to salt are accompanied by increases in intake, and decreases in sensitivity by decreases in intake of salt. These changes in salt intake appear to be part of the organism's reaction to changes in the sodium concentration of the extracellular fluid and would assist in maintaining isotonicity of the body fluids. Further work is required to elucidate the nature of (1) the mechanism responsible for the changes in sensitivity to salt, and (2) the mechanism responsible for the changes in intake of salt which appear to accompany the changes in sensitivity.

Hartridge (1945), Young (1938, 1941, 1949), Richter *et al.* (1938) and Richter (1942) have advanced hypotheses to explain the first of these problems. They suggest that the changes in taste sensitivity are directly due to the influence of the lowered blood content of the needed substance (i.e. salt) on the taste receptors. For example, Richter (1942) writes: "The processes involved in the selective activity of the rat can be formulated as follows, using the salt appetite of the adrenalectomized rat as an example. In such animals there is a sodium deficiency in all the tissues of the body, including the taste buds of the tongue; consequently a salt solution of a given concentration brought into contact with the taste buds in such an animal would encounter a different situation from that found in the normal rat, with the higher salt content. In particular, the sodium ion would be expected to diffuse through the membrane more readily and thus stimulate the taste buds at a lower concentration" (p. 74).

Richter's statement is somewhat misleading in that it implies that the salt losses following adrenalectomy are more or less evenly distributed throughout the body. This is not so. As salt losses occur, sodium is lost from the extracellular fluid with a consequent lowering of the osmotic pressure which is partly counteracted by a reduction in volume of the ECF up to a point where volume is no longer sacrificed in the interests of maintaining the osmotic pressure, which then falls permanently if the salt losses continue. As hypotonicity of ECF develops there is some movement of water from this compartment into the intracellular fluid. In so far as there is a lowering of the osmotic pressure of the ICF it may be said that there is a slight decrease in the concentrations of potassium, magnesium, sodium, and other constituents of the ICF, but there does not appear to be any loss of sodium from the ICF to the ECF. Under these circumstances there is no justification for saying that "in *particular* the sodium ion. . . ." However, it could be that because of the lowered osmotic pressure of the ICF, the sodium ion, among other ions, may more readily diffuse through the cellular membrane. There is a further unverified assumption in Richter's statement, namely that the sodium ion is *the* stimulating agent for the salt taste!

However, Pfaffmann and Bare (1950) did not find this to be the case. They determined the strength of the concentration of sodium chloride solutions just necessary to elicit an afferent impulse in the chorda tympani nerve of sham adrenalectomized and bilateral adrenalectomized rats. For both groups the critical concentration was at approximately the same value. They concluded "that salt deficiency does not alter the sensitivity of the taste receptors" (p. 324).

It is interesting to note that in the present study and in the salt depletion experiment the changes in sensitivity to salt took place when there were changes in the total osmotic pressure of the body. These changes in osmotic pressure would undoubtedly be accompanied by some change in the physicochemical composition of the taste cells. Within the limits that they occurred, these changes did not appear to affect the sensitivity of the taste receptors for sweet, sour or bitter. Further work, in which the nature and the extent of the changes in the taste cells are investigated, is necessary before the implications of this finding for theories of taste cell stimulation can be worked out.

From the results of this and earlier studies (Yensen, 1959*a*,*b*) on taste it appears that taste sensitivity is influenced by certain changes in the physiological state of the organism. Hence the assumption, often made in studies involving taste, that the stimulus properties of a given taste substance are relatively constant is not correct. For the constancy of the stimulus properties of a given stimulus is dependent not only on invariance in the stimulus itself, but also on a lack of variation in the sensitivity of the receptor mechanism concerned. Since certain changes in the physiological state of the organism appear to influence taste sensitivity, it is essential that these variables (i.e. time of day, dietary intake and salt and water balances) be controlled in studies involving taste.

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SHORTER ARTICLES AND NOTES

SIZE CONSTANCY EFFECT DURING ANGULAR AND RADIAL ACCELERATION

BY

T. C. D. WHITESIDE and F. W. CAMPBELL

From the Institute of Aviation Medicine, Farnborough and Physiological Laboratory, Cambridge

Gregory, Wallace and Campbell (1959) have referred to experiments on apparent change of size of after-images carried out in the human centrifuge. Two points dealing with these experiments call for further clarification.

First, the acclerations to which one is subjected in a centrifuge are principally radial, whilst angular accelerations play a relatively small part, especially if the head is fixed. Thus the "3*g*" acceleration which the subjects experienced was a linear and not an angular acceleration.

The second point is that the sensation of "tumbling" which is experienced when the centrifuge begins to slow down towards the end of a run, is caused in part by the angular deceleration of the arm of the centrifuge and in part by the gondola swinging down towards its vertical stationary position. This displaces the head with reference to the plane of rotation and it is this change in head position which increases stimulation in one pair of semicircular canal receptors whilst decreasing the stimulation in another pair. The position of the subject in this particular experiment resulted in a tumbling sensation forwards. It was only during this stage of the experiment, as pointed out by the authors, that the after image appeared to increase in size.

Since the tumbling sensation is caused by stimulation of the labyrinth, the associated reflexes of vertical nystagmus and of postural responses may well have played a part in the apparent alteration in size of the after image, for the involuntary postural change associated with the sensation of tumbling forwards is to throw oneself backwards. This reflex response would be associated with movement backwards of the head, and may thus be directly responsible for the observed results in this, as in other experiments where the head was moved backwards voluntarily.

We may conclude that an increase in linear acceleration applied to a subject in the centrifuge is not in any way associated with variations in size of the after-image providing changes in head position relative to the plane of rotation are excluded.

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CROWN, S., M.A., Ph.D., 27, Circus Road, London, N.W.8.
DAVIS, R., M.A., D.Phil., Institute of Experimental Psychology, Oxford.
DEUTSCH, J. A., M.A., Ph.D., Institute of Experimental Psychology, Oxford.
DREW, Prof. G. C., M.A., Department of Psychology, University College, London.
ELITHORN, A., M.A., M.B., B.Ch., M.R.C.P., D.P.M., Neurological Research Unit, National Hospital, Queen Square, London.
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FOSS, B. M., M.A., Department of Psychology, Birkbeck College, London.
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GIBBS, C. B., B.Sc., Applied Psychology Research Unit, Cambridge.
GREGORY, R. L., M.A., Psychological Laboratory, Cambridge.
GRINDLEY, G. C., B.Sc., M.A., Psychological Laboratory, Cambridge.
HALL, Prof. K. R. L., M.A., D.Phil., Department of Psychology, University of Bristol.
HALLIDAY, A. M., B.Sc., M.B., Ch.B., M.R.C. Neurological Unit, National Hospital, Queen Square, London.
HARPER, R., M.Sc., Ph.D., Department of Psychology, University of Leeds.
HARRIS, A. D., M.R.C.S., L.R.C.P., Department of Experimental Psychiatry, Medical School, Birmingham.
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HICK, W. E., M.D., Psychological Laboratory, Cambridge.
HIMMELWEIT, Mrs. H. T., M.A., Ph.D., London School of Economics, Houghton Street, London.
HINDE, R. A., B.Sc., M.A., D.Phil., Ornithological Field Station, Madingley, Cambridge.
HOTOPH, N., M.A., London School of Economics, Houghton Street, London.
HOWARD, I. P., B.Sc., Department of Psychology, University of Durham.
HOWARTH, C. I., B.Sc., B.A., D.Phil., Department of Psychology, University of Hull.
HUNTER, I. M. L., B.Sc., D.Phil., Department of Psychology, University of Edinburgh.
HURWITZ, H. M. B., B.A., Department of Psychology, Birkbeck College, London.
JAMES, P. H. R., M.A., Ph.D., Department of Psychology, Queen's University, Kingston, Ontario.
JEEVES, M. A., B.A., Ph.D., Department of Psychology, University of Adelaide.
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KELVIN, R. P., B.A., Ph.D., Department of Psychology, University College, London.
LEONARD, J. A., B.A., Ph.D., Applied Psychology Research Unit, Cambridge.
MACKAY, D. M., B.Sc., Ph.D., Department of Physics, King's College, London.
MACKWORTH, N. H., M.B., Ch.B., Ph.D., Defence Research Medical Laboratories, Toronto 12, Canada.

MacNeill, Miss F. E., M.A., B.Ed., Ph.D., Department of Psychology, University of Manchester.
Newson, L. J., B.Sc., Ph.D., Department of Psychology, University of Nottingham.
O'Connor, N., M.A., Ph.D., M.R.C. Social Psychiatry Unit, Maudsley Hospital, London.
Oldfield, Prof. R. C., M.A., Institute of Experimental Psychology, Oxford.
Pickford, Prof. R. W., M.A., D.Litt., Ph.D., Department of Psychology, University of Glasgow.
Piercy, M. F., M.A., Department of Experimental and Clinical Psychology, Barrow Hospital, Barrow Gurney, nr. Bristol.
Pleydell-Pearce, I., B.A., Ph.D., Department of Psychology, University of Bristol.
Poulton, E. C., M.B., B.Chir., M.R.C.S., L.R.C.P., Applied Psychology Research Unit, Cambridge.
Reid, R. L., M.A., Department of Psychology, University of Aberdeen.
Russell, Prof. R. W., M.A., Ph.D., D.Sc., Department of Psychology, Indiana University, U.S.A.
Russell Davis, D., M.D., M.R.C.P., D.P.M., Medical Psychology Laboratory, Cambridge.
Semeonoff, B., M.A., Ph.D., B.Ed., Department of Psychology, University of Edinburgh.
Summerfield, A., B.Sc., Department of Psychology, University College, London.
Sutherland, N. S., M.A., Ph.D., Institute of Experimental Psychology, Oxford.
Symons, J. R., B.A., Ph.D., Department of Psychology, University of Aberdeen.
Szafran, J., M.A., Ph.D., Department of Psychology, University of Exeter.
Trist, E., O.B.E., M.A., Institute of Human Relations, 2, Beaumont Street, London.
Uttley, A. M., B.Sc., Ph.D., National Physical Laboratory, Teddington.
Venables, P., B.A., Ph.D., M.R.C. Unit for Research in Occupational Adaptation, Maudsley Hospital, London.
Vernon, Prof. M. D., M.A., Sc.D., Department of Psychology, University of Reading.
Vince, Miss M. A., M.A., Psychological Laboratory, Cambridge.
Vowles, D., B.Sc., D.Phil., Department of Psychology, University of Reading.
Watson, A. J., M.A., Psychological Laboratory, Cambridge.
Weiskrantz, L., B.A., B.Sc., Ph.D., Psychological Laboratory, Cambridge.
Welford, Rev. A. T., M.A., Psychological Laboratory, Cambridge.
Whitfield, J. W., M.A., Department of Psychology, University College, London.
Wilkinson, R. T., B.A., Ph.D., Applied Psychology Research Unit, Cambridge.
Zangwill, Prof. O. L., M.A., Psychological Laboratory, Cambridge.

BOOK REVIEWS

Personality and Motivation Structure and Measurement. By R. B. Cattell. Londo Harrap. 1958. Pp. xxiv + 948. 55s.

Cattell presents here the fruits of some thirty years' work in the field on personalit and impresses from the start by sheer weight. As an example of what can be achieve by pertinacity and organization, this book commands one's admiration, the amount effort and patience which must lie behind his nine hundred and fifty pages is staggerin

His work is impressive, too, as exhibiting very clearly the principle that th systematic use of machines in computation makes operations possible on a scale whic was unthinkable without them. It is to Cattell's credit that he has grasped and take advantage of these facilities on a magnificent scale.

The reviewer finds Cattell's behaviourism uncongenial and ultimately inappropriat in this field; but this does not prevent him from recognising that the book contains muc that is of value, so much indeed that no attempt is made in this review to deal wit more than certain selected points. Four issues in particular are specially interesting t experimental psychologists, and are well worthy of their consideration; they are th theme of the three media of personality observation, the distinction between Q and Q data, the concept of T-data, with particular reference to the requirement that the subject be not informed of the method of scoring and finally the effects, on subjects and th information derived, of very extensive enquires.

The three media of personality observation yield L-data, Q-data and T-data, respec tively. The first are drawn from observation of behaviour in a naturally occurring lif situation, "made *in situ*"; the second are based on anwsers to questionnaires, while th third are derived from objective tests. Cattell makes a strong case that all three kind of information are necessary for a full understanding of personality, and also that result obtained from the three media must be integrated. This classification of sources c information runs very close to the requirements of experimental psychology that informa tion should always be obtained by three routes, by observation of the subject, by th subject's own contribution in the form of comment or introspection and by objectiv record of performance. Co-ordination of the three such records lead to very impressiv results, giving vivid pictures of a situation, but it is almost equally important, for experi mental work, to recognize that, since the three records all apply to the same situation valuable information is also to be derived from instances where they do not obviousl complement one another. In Cattell's work, there is however, an important differenc that, while the experimental psychologist can collate the three kinds of information o one and the same subject in one and the same situation, Cattell *cannot do the same wit his data*, because they are by definition derived from different situations. Cattell tell us that what has finally made the publication of this book possible is that material drawn from the three different sources of data have yielded dimensional systems con firming one another. This claim is of first class importance and it is interesting to se how it is developed and sustained in the text. It is at this stage, however, that Cattel is least convincing. While a good many names are common to the list of factors derive from L-data and Q-data, when it comes to naming the factors derived from T-data, it i difficult to find the justification for Cattell's claim. Cattell himself says (p. 832), "On should especially mistrust any naming system which attempts prematurely to assimilat these factors, by psychological intuition alone, to the previous L and Q factors, befor interfactor factorizations have actually been checked. Except in a few instances, the L an Q titles cannot reliably be carried across." This leaves us in an unsatisfactory position and it brings out with clarity and force a principle, which has been raised in connectio with factor analysis again and again: that you get out what you put in. Cattell assure us that his objective tests are based on L and Q results. The more comprehensively therefore, he deals with people, the more incredible it becomes if his T results are *no* related to the L and the Q results. What the reader wants to know, and what is fo Cattell to tell him, is how his list of T factors is related to the lists of L and Q factors

Turning next to the distinction between Q- and Q′-data, we find that while both refer to answers derived, broadly speaking, by questionnaire methods, Q-data are treate simply as behaviour. The data are, on the other hand, designated Q′-data when the are regarded as evidence of internal or psychological states, or are "accepted as state ments about the subject and his consciousness." Cattell devotes a good deal of space t

valuable discussion of the relationship between the two. He brings out the dependence
Q′-data on the sincerity of the subject and coins the useful term "motivational
istortion", but Q-data scores, even when we are aware of the risks, remain regrettably
motivation-situation sensitive."

In fact, Cattell never uses Q′-data but works throughout with Q-data. There is no
eason why he should not, since the procedure of factor analysis works in terms of correla-
ons between scores on test-items, the names and meanings of which are not involved
any stage of the computations. At the end, however, of this analysis, Cattell not
nly names his factors (so many of the names being deliberate neologisms, problems of
eaning are largely avoided), but goes on to give examples of the items which are most
eavily loaded with these factors. By thus using the meaning of the items in the
escription of the factors, he must be assuming that the items have meaning for himself
nd his readers. If he really means, as he says, that "no dependence is placed on the
rdinary meaning of language," then there ought to accompany the tables of items, which
re given with the factor names, the special meanings which as he says, "must be deter-
ined by experiment." It is by no means clear what, then, happens if the items mean
omething different to Cattell and to his subjects.

The distinction between Q and Q′ is nevertheless an important one and one which
not always recognized in work with questionnaires.

A similar point arises in connection with T-data, and here again the issue is one which
xperimental psychologists neglect at their peril. T-data are derived from objective
sts; the definition given here of such tests is somewhat narrow, but contains the
nportant provision that the subject shall not be informed as to how the test is scored.
o put the matter another way, the subject is "told what he should try to do but not
hat this will be taken to mean about him in terms of his self concept." It is easy
nough for Cattell to adopt this position and to claim that his subjects act without informa-
on, but it is perfectly obvious to anyone who has conducted psychological experiments
at a subject nearly always has ideas of his own as to what his performance is for, and
at his ideas can materially affect his performance. Cattell has introduced the concept
motivational distortion for questionnaires, and it seems almost unbelievable that he
ould not be aware of the importance of this factor for objective test results, whether
e talks of motivational distortion or of the "private instructions," in terms of which
subject carries out a test, however straightforward and explicit the public instructions
ay seem to be. It is just at this point that co-ordination of the three streams of
formation, referred to above, could be so informative and where Cattell seems to have
scarded or shut his eyes to such crucial material. It is worth remarking that Thurstone
ew attention to the risk of subjects misunderstanding questions or interpreting them
a way not envisaged by the investigator.

Cattell dismisses a number of such difficulties by claiming that he was working
roughout with "normal co-operative subjects." It is legitimate for Cattell not to be
ncerned, at this stage, with the problem of producing methods and material which
uld be applied to ordinary people in ordinary life situations. It is not unreasonable
attempt to tackle the problems of personality and motivation on a vast laboratory
ale and it is just possible to suppose that this is comparable to the study of behaviour
physical objects *in vacuo*. What is more difficult to suppose is that observations, on
ch a scale as is clearly indicated in this book, can possibly proceed in such a way as
t to produce, in the subject (and in the observer), an entirely special and artificial
tlook and set of responses. It looks as if the power of the machines at Cattell's
sposal may well outrun the power of the human being to respond or observe. Another
ature of the virtually unlimited capacity of the laboratory to handle data is to be seen
the specification equations which can be elaborated in relation to any performance.
these lengthen out to contain more and more variables, their meaning becomes cloudy
r the human interpreter. One can envisage the situation where the specification
uations turned out by one computing machine are, to adapt the story of the old lady's
estion about the sex of a hippopotamus, only of interest to another computing machine.

Beyond these four issues, two others may be selected, for comment, from a very
rge "parent population," for instance, as is admitted by Cattell, specification equations
hich only treat factors additively may well not be adequate to express their interactions.
is clear from the discussion that various complexities of interaction between factors
n be accommodated by introducing product terms into the specification equations.
this connection he makes the important assertion that the manner in which factors
mbine mathematically corresponds to the way in which they combine psychologically.

While this may conceivably be held of factors which have themselves been *derived* mathematically, it is by no means obvious that we can extrapolate such a "correspondence" to the psychological situations and influences of human experience before they have been through the mills of behaviourism and factor analysis.

Almost alone among factor analysts, Cattell has made much of changes over periods of time, and his proposed P-technique is an attempt to take care of this very important aspect of personality. He claims that this method would allow changes through time to become apparent in the factor structure, not only if the tests were specially constructed, but even if ordinary tests were used repeatedly. His examples are quite striking, but once again the effort involved for the subject seems likely to be a bar to wider application of this method.

Cattell deals with an enormous range of material and method with masterly assurance and amateurs may well shrink from commenting on the validity of the computational methods he has devised. The most difficult problem facing the reader will be to determine what is, after all, central; namely, the value of the system of factors of which Cattell is now compiling a Universal Index. It is a problem which this writer will not offer to solve, beyond saying that the chapters in which the factors are described and deployed are the ones least likely to be of interest to those who are not already confirmed in their acceptance of factor analysis.

B. Babington Smith.

Ageing and Human Skill. By A. T. Welford. London. Oxford University Press. 1958. Pp. ix + 300. 25s.

This book respresents the results of a 10-year search for an understanding of the mechanisms of skill and of ageing—the work of the Nuffield Unit for Research into Problems of Ageing—which was carried on under the direction of the author. It reports and evaluates the experimental findings from numerous original studies conducted by the staff of the Unit, and puts these experimental data into a substantial theoretical framework which is based on an evaluation of a wide variety of studies from other sources.

The author is at his best in Chapter 2, where he writes about the nature of skill, and in Chapter 6, where he develops the concept of translation processes in skills. He feels that skilled activity has three essential characteristics—it is organized, learned, and serially patterned. Welford's treatment of skill is therefore very similar to that developed by Sir F. C. Bartlett in his recent book on *Thinking*. Sensory and perceptual process, problem solving, learning and memory, and adaptation are treated in separate chapters but consistently from the viewpoint of skill. As an illustration, learning how to learn is viewed as an aspect of the development of skill.

The importance of central (as contrasted with peripheral or motor) processes is emphasized throughout the book. Thus it is concluded that the ". . . slowing of sensori-motor performance with age is due not to longer time required to execute movements *as such*, but to longer time needed to initiate, guide, and monitor them, owing to a limitation in the capacity of central processes" (p. 107). Eleswhere, it is concluded that ". . . the difficulty for older people in many sensori-motor tasks lies in what we have termed translation processes" (p. 129), that the ". . . difficulty of visual perception in later middle and old age is not solely due to readily observable and commonly recognized changes in the eye" (p. 190) and that ". . . changes with age lie essentially in the central control and guidance of actions" (p. 254).

One of the broadest integrating ideas used to account for skill changes is the older man's decreasing capacity for short term (immediate or running) memory. The analogy can be drawn to a limited buffer storage capacity in a high speed computer; older people are found to experience increasing difficulty in handling speeded or machine-paced tasks, perceptual-motor tasks in which there are occasional short periods of information overload, or problem-solving tasks in which new and different pieces of data must be related.

The systematic treatment of ageing is not particularly novel. Rather it is characterized by attempts to evaluate commonly held ideas, such as slowing of performance, loss of adaptability, and slowness in learning new tasks, by examining such concepts critically in the light of the latest experimental evidence. The result is a balanced treatment which develops several rather broad concepts. Skill capacity is shown to change in many ways with age; these changes cannot be eliminated or reversed, but a deeper understanding of skill and ageing may suggest many ways whereby, from a practical standpoint, workers can be helped to adjust to them and industrial jobs can

be changed so as to minimize handicaps to the older worker. The chapters on speed and accuracy of movement, and on pacing accidents and heavy work provide the greatest amount of new data on ageing. Again they offer no panacea that will solve the problems of the older worker, but they do leave a clear picture of constructive steps that can be taken to adapt jobs to the worker such as alleviation of speed stress, and provision for self regulation of the pace of work. In these chapters and elsewhere experiments conducted within industry are reported along with findings from the experimental laboratory, adding considerably to the generality of the results.

There are, of course, limitations to the data on skill and on ageing changes. Cross-sectional comparisons are recognized to be less desirable, from the viewpoint of experimental design, than longitudinal studies, but apparently no long-term studies were undertaken by the Nuffield Unit. Chapters that cover topics that are commonly not considered under skill, such as perception and problem solving, in general contain less original research data than do chapters on such topics as speed and accuracy of movements.

Everying considered, this is a thoroughly excellent book. It should be in the library not only of those who are interested either in skill or in ageing, but in systematic psychology as well.

PAUL M. FITTS.

Instinctive Behaviour. Contributors: Konrad Lorenz, D. J. Kuenen, Nicholas Tinbergen, Paul Schiller, Jacob von Uexkull. Translated and edited by Claire H. Schiller. Introduction by Karl S. Lashley. London. Methuen. 1958. Pp. 328. 45s.

These essays, all but one of which were published originally in German, are collected here in translation as representing "the development of a modern concept" of instinctive behaviour. The first, a long paper by von Uexkull, represents a point of departure—it emphasizes that an animal's perception of its environment depends on its sensory capacities. This was an important conclusion at the time, and the paper contains many interesting observations, as well as what now seem some rather far-fetched speculations. The bulk of the book consists of six essays by Lorenz, Tinbergen and Kuenen, alone or in combination, including Lorenz's (1935) "Kumpan" paper and the classic papers on the egg-rolling of the Greylag goose and the begging of young thrushes. It will surprise many to discover how nearly all the ideas which have proved useful in ethology were present in fully-fledged form in these essays written 20 years ago. The use of fixed action patterns in comparative study, the evolution and function of social releasers, the nature of animal language, the integration of discrete actions into functional sequences of behaviour, and many other topics are discussed here in detail; and the nature of Lorenz's and Tinbergen's genius is shown by the fact that later workers on such subjects have done little more than fill in the details. At the same time, these papers contain much of the earlier theoretical background to ethology which is now discarded by most ethologists as well as by Lorenz and Tinbergen themselves. Lorenz's discussions of the innate/learning controversy, releasing mechanisms or the precise nature of "action-specific energy" make curious reading now; and it would be a mistake to take these essays as at all representative of their views to-day.

Many of these earlier ideas were crystallized in Tinbergen's hierarchical scheme of nervous centres, and the last essay by Lorenz in this book (1952) contains a summary of this model. From this point Tinbergen's thinking entered a new phase, the beginning of which can be seen in the 1954 Macy Foundation volume on Group Processes, where many aspects of ethological work are discussed and the distinction between the invaluable general principles on which ethological work is based on the one hand, and the rather loose theoretical concepts on the other, becomes apparent.

This volume was planned and begun by the late Paul H. Schiller. It contains one previously unpublished essay by him on the importance of "innate" organization in determining the course of learning in Chimpanzees, in which he emphasizes that a knowledge of this organization is essential for analysing the acquisition of skills. This paper was ahead of its time in pointing the way in which ethology is likely to develop—by establishing bridgeheads with other fields of study in which ethological observations and methods can be applied.

R. A. HINDE.

INDEX OF AUTHORS

ANNETT, J. - - - - - - - - - - - - - - 3
BEGBIE, G. H. - - - - - - - - - - - - - 65
BENTON, A. L. - - - - - - - - - - - - - 39
BERTELSON, P., with Davidson, A. - - - - - - - - 180
CAMPBELL, F. W., with Gregory, R. L. and Wallace, J. G. - - - - 54
CAMPBELL, F. W., with Whiteside, T. C. D. - - - - - - - 249
CHEESMAN, G. H., with Kirby, H. M. - - - - - - - - 115
CLARKSON, J. K., with Deutsch, J. A. - - - - - - - 143 & 150
DAVIDSON, A., with Bertelson, P. - - - - - - - - - 180
DAVIS, R. - - - - - - - - - - - - - - 211
DEUTSCH, J. A. - - - - - - - - - - - - - 155
DEUTSCH, J. A., with Clarkson, J. K. - - - - - - - 143 & 150
ETTLINGER, G. - - - - - - - - - - - - - 164
GLASER, F., with Leibowitz, H., Waskow, I and Loeffler, N. - - - - 108
GREGORY, R. L. - - - - - - - - - - - - - 113
GREGORY, R. L., with Wallace, J. G. and Campbell, F. W. - - - - 54
GROSS, C. G., with Weiskrantz, L. - - - - - - - - - 49
HOWARTH, C. I., with Treisman, M. - - - - - - - - - 129
KHAIRY, M. - - - - - - - - - - - - - - 84
KIRBY, H. M., with Cheesman, G. H. - - - - - - - - 115
LEIBOWITZ, H., with Waskow, I., Loeffler, N. and Glaser, F. - - - 108
LEONARD, J. A. - - - - - - - - - - - - - 76
LOEFFLER, N., with Leibowitz, H., Waskow, I. and Glaser, F. - - - 108
MORAY, N. - - - - - - - - - - - - - - 56
MOWBRAY, G. H., with Rhoades, M. V. - - - - - - - - 16
O'CONNOR, N., with Venables, P. H. - - - - - - - - 175
OSWALD, I. - - - - - - - - - - - - - - 45
RHOADES, M. V., with Mowbray, G. H. - - - - - - - - 16
SANDSTRÖM, C. I. - - - - - - - - - - - - - 33
SUTHERLAND, N. S. - - - - - - - - - - - - - 24
TREISMAN, M., with Howarth, C. I. - - - - - - - - 129
VENABLES, P. H., with O'Connor, N. - - - - - - - - 175
WALLACE, J. G., with Gregory, R. L. and Campbell, F. W. - - - - 54
WASKOW, I., with Leibowitz, H., Loeffler, N. and Glaser, F. - - - - 108
WASON, P. C. - - - - - - - - - - - - - 92
WEISKRANTZ, L., with Gross, C. G. - - - - - - - - 49
WELFORD, A. T. - - - - - - - - - - - - - 193
WHITESIDE, T. C. D., with Campbell, F. W. - - - - - - - 249
YENSEN, R. - - - - - - - - - - - - - 221, 230 & 239

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HANS JÜRGEN EYSENCK, Das "Maudsley Personality Inventory" als Bestimmer der neurotischen Tendenz und Extraversion.

WALLACE A. RUSSEL und OSKAR R. MESECK, Der Einfluss der Assoziation auf das Erinnern von Worten in der deutschen, französischen und englichen Sprache.

FRANZ WEINERT, Der Einfluss der Übung auf das aufgabenbezogene Verhalten bei 10 jährigen Kindern.

HEINZ SCHMIDTKE, Analyse des Arbeitsablaufes unter besonderer Berücksichtigung der Arbeitspausen.

EVA GRUHNWALD und EBERHARD ULICH, Über die Anwendung des Arbeitsversuches bei psychisch Beeinträchtigten unter besonderer Berücksichtigung einer Untersuchung an taubstummen Jugendlichen.

WERNER TRAXEL und GUNHILD WREDE, Hautwiderstandsänderungen bei Musikdarbietungen.

ELFI ZEBLEWSKI, Die Anwendbarkeit des Perseverationstests von Mittenecker für die Persönlichkeitsdiagnostik.

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CONTENTS

A. T. WELFORD. Evidence of a Single-Channel Decision Mechanism Limiting Performance in a Serial Reaction Task - 193

R. DAVIS. The Role of "Attention" in the Psychological Refractory Period - - - - - - - - - - - - - 211

ROY YENSEN. Some Factors Affecting Taste Sensitivity in Man. I: Food Intake and Time of Day - - - - - - - 221

ROY YENSEN. Some Factors Affecting Taste Sensitivity in Man. II: Depletion of Body Salt - - - - - - - - - 230

ROY YENSEN. Some Factors Affecting Taste Sensitivity in Man. III: Water Deprivation - - - - - - - - - 239

Shorter Articles and Notes

T. C. D. WHITESIDE and F. W. CAMPBELL. Size Constancy Effect During Angular and Radial Acceleration - - - - 249

Experimental Psychology Society: Founder Members - - - 250

Book Reviews - - - - - - - - - - - - 252

Index of Authors - - - - - - - - - - - - 256

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